"A STUDY ON THE CLINICAL PROFILE,DIAGNOSTIC WORK UP AND FOLLOW UP OF CHILDREN AGED 2 MONTHS TO 12 YEARS WITH THROMBOCYTOPENIA"

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TIRUNELVELI MEDICAL COLLEGE HOSPITAL

THE TAMIL NADU DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI ,TAMIL NADU

CERTIFICATE

This is to certify that the Dissertation entitled **"A STUDY ON THE CLINICAL PROFILE,DIAGNOSTIC WORKUP AND FOLLOW UP OF CHILDREN (2 MONTHS TO 12 YEARS) WITH THROMBOCYTOPENIA"** submitted by **Dr.R.JEGAN MURUGAN** to The Tamilnadu Dr. M.G.R. Medical University, Chennai, in partial fulfilment for the award of M.D.Degree(Paediatrics) is a bonafide work carried out by him under my guidance and supervision during the academic year 2010-2013. This dissertation partially or fully has not been submitted for any other degree or diploma of this university or other.

Prof.Dr.LAKSHMI. MD.,
Unit Chief, UNIT II,
Department of Paediatrics,
Tirunelveli Medical College ,
Tirunelveli – 627011.

Prof. Dr.S .DEVIKALA,MD.,
Professor and HOD,
Department of Paediatrics,
Tirunelveli Medical College,
Tirunelveli – 627011.

The Dean,
Tirunelveli Medical College,
Tirunelveli – 627 011.

DECLARATION

I**, Dr.R.JEGAN MURUGAN**, solemnly declare that the Dissertation titled **"A STUDY ON THE CLINICAL PROFILE,DIAGNOSTIC WORKUP AND FOLLOW UP OF CHILDREN(2 MONTHS TO 12 YEARS) WITH THROMBOCYTOPENIA"** has been prepared by me.

This is submitted to the Tamilnadu Dr.M.G.R. Medical University, Chennai, in partial fulfillment of the regulations for the award of MD Degree Branch VII (PAEDIATRICS).

It was not submitted to the award of any degree/diploma to any University either in part or in full previously.

Place: TIRUNELVELI

Date:

DR.R.JEGAN MURUGAN,
POST GRADUATE,
M.D.PAEDIATRICS,
TIRUNELVELI MEDICAL
COLLEGE HOSPITAL

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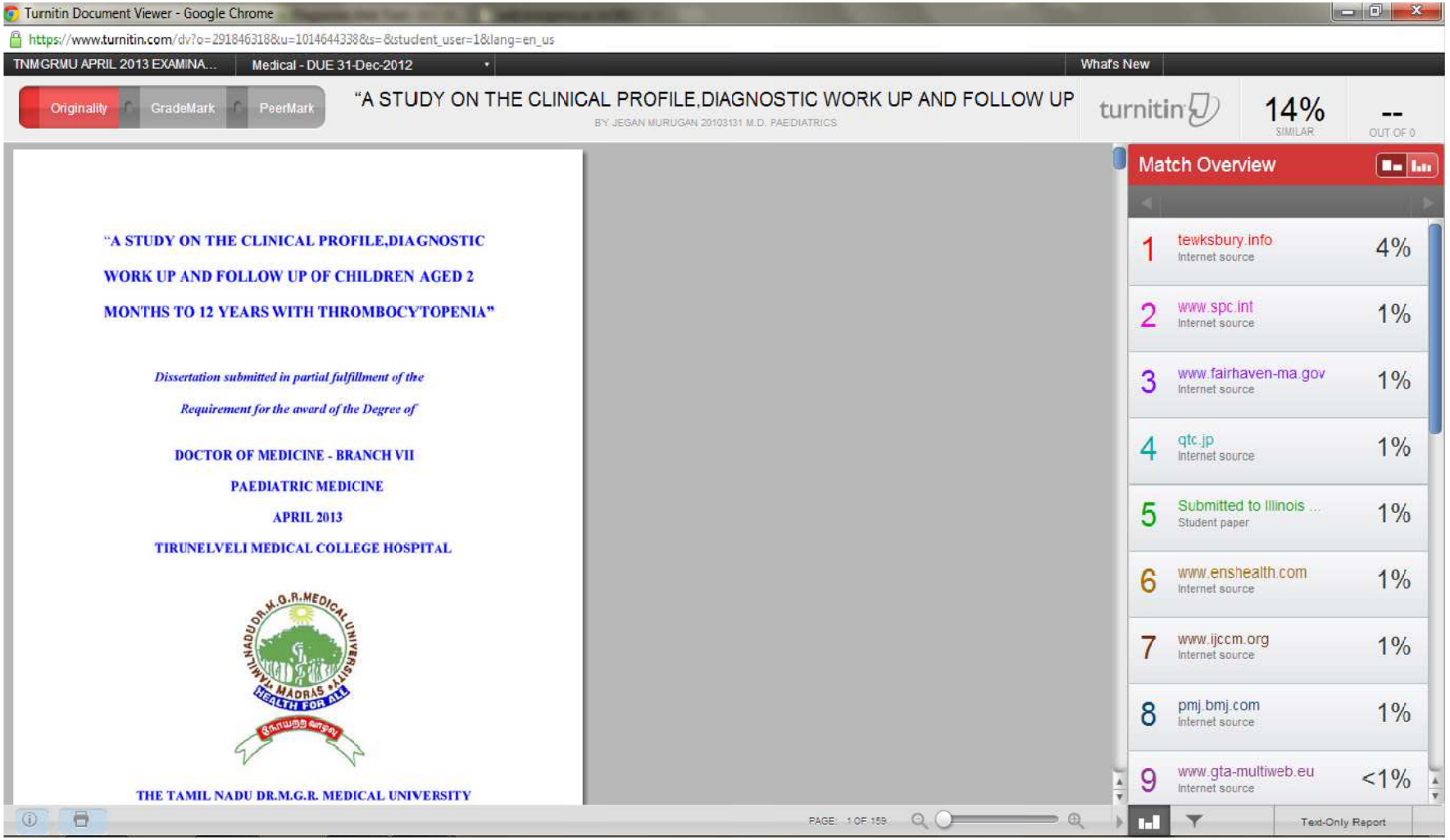

TIRUNELVELI MEDICAL COLLEGE

TIRUNELVELI,

STATE OF TAMILNADU, INDIA

PIN CODE:627011

Tel: 91-462-2572733, 2572734 Fax: 91-462-2572944

Estd:1965

Under the Directorate of Medical Education, Government of Tamilnadu.

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Certificate of Approval

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Ethical Committee,
Tirunelveli Medical College,
Tirunelveli-11.

CONTENTS

INTRODUCTION

1. INTRODUCTION

Normal hemostasis is not only a complex but also an ingenious system which maintains blood in the vascular system free from clots,the vital element of the process being the platelet[1].Decreased platelet count is not as common as anemia,the hematological cousin. Literature quotes the incidence of thrombocytopenia to vary from 13 to 58% in various studies[2].But it is far more dangerous and resource consuming to the emergency department and the ICU setting.It can be associated with bleeding ranging from minor bleeds to life threatening intracranial hemorrhage.

There is very often a poor correlation between the extent of thrombocytopenia and the severity of the bleed.Guidelines on platelet transfusions are also varied and confounding.Hence the treatment of thrombocytopenia has to be guided by an understanding of the cause and clinical course.It is often said that the main treatment goal in all patients with decreased platelet count is to maintain a safe platelet level so as to prevent significant bleeding.But what constitutes a safe count in a specific patient varies, depending on the etiology of the thrombocytopenia as regards to whether it is transient or chronic, as well as the patient's expected level of disease activity[3].

There have been a plethora of studies on anemia and its impact on various disease processes.But thrombocytopenia is still a grey area waiting to be explored.Again,there are lot of studies in the adult population detailing the outcome of patients with thrombocytopenia in the Intensive care setting[4].But similar studies in the paediatric age group are lacking. No particular study has been addressed towards studying the relative frequency of different disease conditions presenting as newly diagnosed thrombocytopenia in paediatric patients presenting to an Indian tertiary care hospital. The need arises to look at thrombocytopenia as a whole and to gather knowledge regarding the common disease entities presenting as thrombocytopenia, the clinical course in the hospital of patients presenting as such and whether or not active treatment modalities like platelet transfusions, steroids, platelets are required in them. This knowledge will give the clinician an idea of approach to a paediatric patient detected to have thrombocytopenia on admission to a tertiary care hospital in India.

The present study was thus undertaken to evaluate the occurrence of thrombocytopenia,assess the cause for it and the associated mortality and morbidity in paediatric patients. In addition,the association with bleeding and the requirement of blood products was also studied.

AIMS AND OBJECTIVES

2. AIMS AND OBJECTIVES

AIMS OF THE STUDY:

To assess the clinical and laboratory profile of patients having thrombocytopenia(<1 lakh) admitted as inpatients in the children medical ward of Tirunelveli Medical College Hospital.

OBJECTIVES OF THE STUDY:

1. To evaluate the relative frequency of different disease conditions presenting as newly found thrombocytopenia and to identify the cause for thrombocytopenia in children.
2. To assess the clinical complications,outcome and prognosis associated with thrombocytopenia,especially the proportion of patients who had bleeding manifestations.
3. To evaluate the percentage of patients requiring interventions like platelet substitutes, steroids,other blood products and to determine whether a low platelet count or presence of bleeding manifestations was considered as an indication for platelet transfusions.

REVIEW OF LITERATURE

3. REVIEW OF LITERATURE

HISTORY OF THROMBOCYTES :

In 1877, Osler coined the term thrombocytes or haematoblasts of Deetjen and Dekhuyzen (1901) and elucidated the role of these third corpuscles as fibrin formers in coagulation.

PLATELETS-BRIEF NOTE ON ROLE IN NORMAL PHYSIOLOGY:

Platelets play a central role in normal hemostasis and therefore also in thrombosis. Despite their lack of nucleus, they (about 2 micrometer in diameter) are amazingly versatile. In the haemostatic cascade platelets undergo 3 important reactions: (1) adhesion and shape change, (2) secretion and (3) aggregation, collectively called to as platelet activation[5].The platelet surface has a number of receptors for adhesive proteins, including von Willebrand factor (VWF) and fibrinogen, as well as receptors for platelet aggregation,like thrombin, collagen and adenosine diphosphate (ADP). After injury to the vessel wall, subendothelial collagen binds VWF. VWF undergoes a conformational change that induces binding of the GPIb complex (the VWF receptor). This is called *platelet adhesion.* Platelets then undergo activation. During activation, the platelets generate thromboxane A_2 from arachidonic acid via cyclo-oxygenase.After activation,agonists such as ADP, ATP, Ca^{2+}, serotonin, and coagulation factors,are released

into the surrounding milieu. Circulating fibrinogen binds to the GPIIb-IIIa complex, linking platelets together.This process is called *aggregation.*

These events lead to the development of a hemostatic plug at the site of vascular injury. The serotonin and histamine that are released during activation increase the local vasoconstriction. In addition to these actions, the platelet also provides the catalytic surface on which clotting factors assemble and eventually generate thrombin.Lastly, the platelet contractile proteins and cytoskeleton play a role in clot retraction[6].

DEFINITION OF THROMBOCYTOPENIA:

There are an estimated 15,000,000 megakaryocytes/kg body weight in the body,each of which produces approximately 1000–1500 platelets[7].Normal platelet count ranges between 1.5 lakhs to 4.0 lakhs per microlitre.Definition of thrombocytopenia is not unequivocal as some books[8] indicate it as less than 100×10^9/L and others as less than 150×10^9/L; but a working definition of less than 1 lakh is widely accepted.Furthermore, it is considered mild (and usually asymptomatic)if above 50×10^9/L, moderate if it is between 30×10^9/L and 50×10^9/L,and severe if the platelet count is below 30×10^9/L.[9]

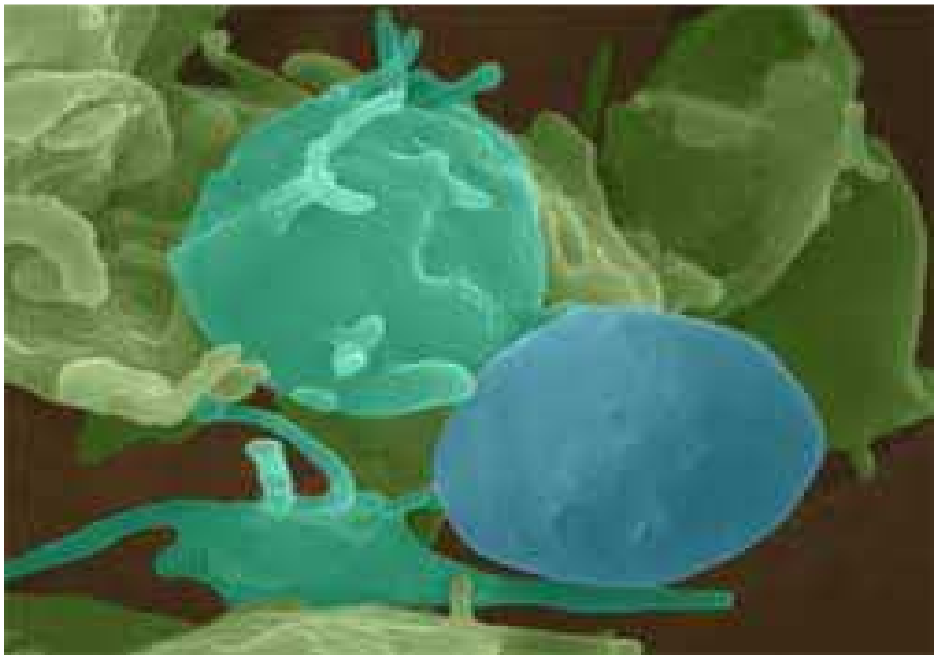


FIGURE 1: ELECTRON MICROSCOPIC IMAGE OF THE PLATELET

FIGURE 2:PETECHIAE AND PURPURA

ETIOLOGY:

The causes of thrombocytopenia include:

1. **DECREASED PRODUCTION** on either a congenital or an acquired basis;
2. **SEQUESTRATION** of the platelets within an enlarged spleen or other organ; and
3. **INCREASED DESTRUCTION** of normally synthesized platelets on either an immune or a nonimmune basis.

TABLE 1:DIFFERENTIAL DIAGNOSIS OF THROMBOCYTOPENIA IN CHILDREN AND ADOLESCENTS:[10]

DESTRUCTIVE THROMBOCYTOPENIAS
Primary Platelet Consumption Syndromes
Immune thrombocytopenias
Acute and chronic ITP(Idiopathic thrombocytopenic purpura)
Autoimmune diseases with chronic ITP as a manifestation
Cyclic thrombocytopenia
Autoimmune lymphoproliferative syndrome and its variants
Systemic lupus erythematosus
Evans syndrome
Antiphospholipid antibody syndrome
Neoplasia-associated immune thrombocytopenia
Thrombocytopenia associated with HIV
Neonatal immune thrombocytopenia

Alloimmune

Autoimmune (e.g., maternal ITP)

Drug-induced immune thrombocytopenia (including heparin-induced thrombocytopenia)

Post-transfusion purpura

Allergy and anaphylaxis

Post-transplant thrombocytopenia

Nonimmune thrombocytopenias

Thrombocytopenia of infection

Bacteremia or fungemia

Viral infection

Protozoal infection

Thrombotic microangiopathic disorders

Hemolytic-uremic syndrome

Thrombotic thrombocytopenic purpura

Bone marrow transplantation–associated microangiopathy

Drug-induced

Platelets in contact with foreign material

Congenital heart disease

Drug-induced via direct platelet effects (ristocetin, protamine)

Type 2B VWD or platelet-type VWD

Combined Platelet and Fibrinogen Consumption Syndromes
Disseminated intravascular coagulation
Kasabach-Merritt syndrome
Virus-associated hemophagocytic syndrome
IMPAIRED PLATELET PRODUCTION
Hereditary disorders
Acquired disorders
Aplastic anemia
Myelodysplastic syndrome
Marrow infiltrative process
Osteopetrosis
Nutritional deficiency states (iron, folate, vitamin B_{12}, anorexia nervosa)
Drug- or radiation-induced thrombocytopenia
Neonatal hypoxia or placental insufficiency
SEQUESTRATION
Hypersplenism
Hypothermia
Burns

This can be further simplified and considered as follows:[11]

1. Pseudothrombocytopenia
2. Congenital thrombocytopenia
3. Acquired thrombocytopenia
    a. Platelet sequestration
        - - Hypersplenism
    b. Decreased production
        - - Neoplasia (bone marrow infiltration or cytotoxic drugs)
        - - Viruses (EBV, CMV, rubella, varicella, parvovirus)
        - - Megaloblastic anaemia
    c. Increased destruction
        - - Immune-mediated (ITP, neonatal alloimmune thrombocytopenia, drug-induced ITP, post-transfusion, autoimmune diseases, lymphoproliferative disorders, HIV, HCV and *Helicobacter pylori* infections, HIT
        - - Not immune-mediated (vascular prostheses, DIC, TTP/HUS, HELLP, eclampsia)

This classification certainly allows us to work up with more ease from a clinical point of view in that it allows a differential diagnosis to be made on the basis of symptoms, findings and blood counts.

Out of these disorders listed,few of the common conditions that are encountered in the clinical setting are discussed below with regards to the cause of thrombocytopenia in those conditions and their prognosis and treatment.

I.PSEUDOTHROMBOCYTOPENIA:

Laboratory platelet counts are prone to error. This is due to in vitro agglutination of platelets in blood collected into tubes containing EDTA[12].Hence,a single platelet count that is lower than normal should be always confirmed by a second count. It should also be confirmed by inspecting the blood film[13]. "Platelet satellitism",a phenomenon due to adhesion of platelets to polymorphonuclear cells,can also be excluded by seeing the smear[14].

II.INFECTIOUS CAUSES:

Thrombocytopenia is seen in several infections.It is a common cause of the bleeding seen in infectious diseases[15].

A.VIRUSES:

Mild thrombocytopenia is frequently associated with viral infection[16].Although the pathophysiologic mechanisms are not exactly known, a production deficit is probably cited as important in many of the cases. Megakaryocytes containing inclusion bodies are seen in dengue, other hemorrhagic fevers, hepatitis,varicella, cytomegalovirus, infectious

mononucleosis, chickenpox and parvovirus infections[17,18].Even live measles virus vaccination can also cause thrombocytopenia[19].

1.DENGUE:

In the Indian setting dengue is one of the commonest causes of transient thrombocytopenia.Though transient,it is often very severe.Associated with the plasma leakage that is characteristic of the disease,it is an important cause of mortality and morbidity in Indian children.INDIA falls under Category A of the WHO South East Asia region epidemiological data review.This includes countries where dengue is a

- Major public health problem
- Hyperendemicity with all four serotypes circulating in urban areas.
- Leading cause of hospitalization and death among children.
- Spreading to rural areas.

In-country geographic expansion is occurring in India and Cyclic epidemics are increasing in frequency [20].

The dengue viruses are members of the genus *Flavivirus* and family *Flaviviridae*. Aedes (Stegomyia) aegypti (Ae. aegypti) and Aedes (Stegomyia) albopictus (Ae. albopictus) are the two most important vectors of dengue.The genome of the dengue virus is composed of seven non-structural protein (NS) genes and three structural protein genes. Among nonstructural proteins, the envelope glycoprotein, NS1, is of diagnostic and

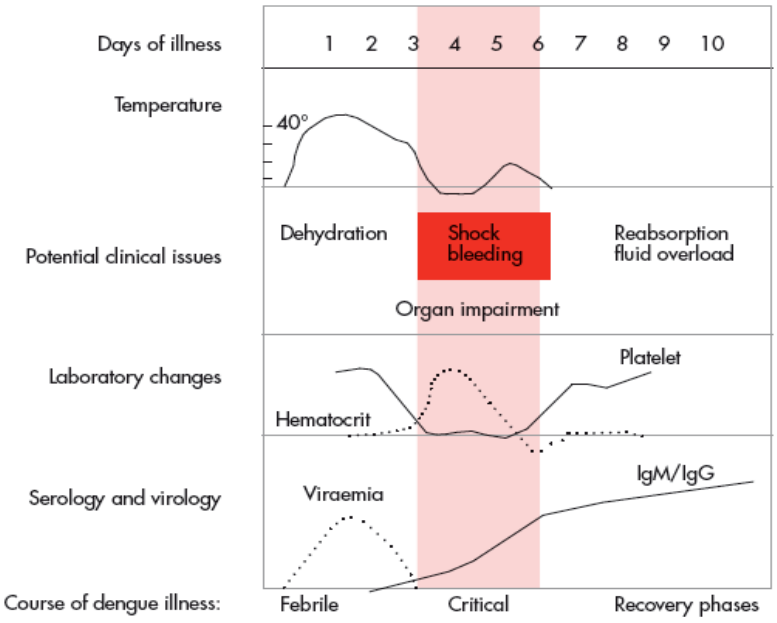
pathological importance. Plasma levels of secreted NS1 (sNS1) correlate with viral titers, being higher in patients with DHF compared with dengue fever[21]. Moreover, elevated free sNS1 levels within 72 hours of onset of illness identify patients at risk of developing DHF. Very high levels of NS1 protein are detected in acute phase samples from patients with secondary dengue infections but not primary infections. This suggests that NS1 may contribute to formation of circulating immune complexes, which are thought to have an important role in the pathogenesis of severe dengue infections[22].

There are four dengue virus serotypes, which are denoted as DENV-1, DENV-2,DENV-3 and DENV-4. Infection with any one serotype confers lifelong immunity to that virus serotype.Although all four serotypes are antigenically similar, they are different enough to elicit cross-protection for only a few months after infection by any one of them. Secondary infection with another serotype or multiple infections with different serotypes leads to severe form of dengue (DHF/DSS).

Dengue virus infection may be asymptomatic or may cause undifferentiated febrile illness (viral syndrome), dengue fever (DF), or dengue haemorrhagic fever (DHF) including dengue shock syndrome(DSS).

Course of dengue illness is characterized by three phases-febrile phase,critical phase and the recovery phase.As the figure below shows clearly,the manifestations are well documented and can be well anticipated if diagnosis has been made early serologically.

Figure 3:The course of dengue illness



FEBRILE PHASE:

Febrile phase is the initial phase during which dengue presents like any other illness,often goes unrecognized because of the gross similarity with other infections.High grade fever is present which typically lasts 2-7 days and is often accompanied by facial flushing, generalized body ache, myalgia, arthralgia,skin erythema and headache[23].Injected pharynx,sore throat and conjunctival injection may be seen in few patients,as also anorexia, nausea and vomiting.Tourniquet test may be positive in this phase

and positivity increases the probability of dengue[24].Monitoring for warning signs during this phase is vital to recognize progression to the critical phase.

TABLE 2:WARNING SIGNS IN DENGUE

Clinical	Abdominal pain or tenderness Persistent vomiting Clinical fluid accumulation Mucosal bleed Lethargy, restlessness Liver enlargement >2 cm
Laboratory	Increase in HCT concurrent with rapid decrease in platelet count

Petechiae,gum bleeds and epistaxis are the usual mild hemorrhagic manifestations that are seen in this phase .Gastrointestinal bleeding may occur,but is uncommon[25].Progressive decrease in total white cell count, is probably the only early hematological abnormality.

CRITICAL PHASE

On days 3–7 of the illness, an increase in capillary permeability along with increasing hematocrit levels and dropping platelet counts is seen[26].This is the period of defervesence of fever and is the onset of the critical phase,associated with increasing mortality and morbidity.Plasma leakage usually lasts 24–48 hours.

At this point patients without plasma leakage will improve, while those with increased plasma leak become worse because of the lost plasma

volume. Pleural effusion and ascites may be the clinically recognisable evidence of plasma leakage,hence the importance of Chest X-ray and abdominal ultrasound for diagnosis.The degree of increase above the baseline hematocrit is usually more than 20% in DHF and correlates with the disease severity.Lesser degrees of rise are associated with milder manifestations.

Dengue Shock syndrome(DSS) occurs when critical volume of plasma(usually more than 25%) is lost.It is often preceded by warning signs and hence in most cases,early identification is possible with careful monitoring.Disseminated intravascular coagulation may be triggered by prolonged plasma leak and shock. This in turn leads to severe haemorrhage.Hematocrit may thus decrease in severe shock.End organ damage in the form of severe hepatitis, encephalitis or myocarditis and/or severe bleeding may also develop[27].

Serial monitoring of the platelet count and hematocrit is recommended during this phase to identify the onset of recovery phase and to guide fluid therapy.

RECOVERY PHASE:

In the next 48–72 hours,if the patient survives,a gradual reabsorption of extravascular fluid takes place. Appetite returns, general well-being improves, gastrointestinal symptoms abate.Hemodynamic status stabilizes

and diuresis occurs. Some patients may develop a erythematous rash described as“isles of white in the sea of red”[28]. Bradycardia is common during this stage.

The hematocrit may sometimes even be lower due to the dilutional effect of reabsorbed fluid. Soon after defervescence the white blood cell count usually starts to rise but the recovery of platelet count is typically later.

Respiratory distress is likely to occur if excessive intravenous fluids had been administered,because of the development of pulmonary oedema or congestive heart failure.

The exact cause of thrombocytopenia in dengue is unknown,but is thought to be multifactorial; There is enhanced production of anti-platelet and anti-NS1 antibodies in dengue.This is caused by cytokine overproduction(Interleukin-6) induced by the virus.There is also aberrant immune activation.These antibodies cross-react with human platelets[29] .Reduced production of platelets in DF and increased destruction in DHF may also be the reasons [30,31]. The reduction in the counts may also be due to bone marrow suppression induced by the dengue virus [32]. Viral antigen antibody complex induced destruction of circulating platelets has also been documented[33].

TABLE 3:WHO CLASSIFICATION OF DENGUE INFECTIONS AND GRADING OF SEVERITY OF DHF:[34]

DF/ DHF	Grade	Signs and Symptoms	Laboratory
DF		Fever with two of the following: • Headache. • Retro-orbital pain. • Myalgia. • Arthtralgia/bone pain. • Rash. • Haemorrhagic manifestations. • **No evidence of plasma leakage.**	• Leucopenia (wbc ≤5000 cells/mm³). • Thrombocytopenia (Platelet count <150 000 cells/mm³). • Rising haematocrit (5% – 10%). • No evidence of plasma loss.
DHF	I	Fever and haemorrhagic manifestation (positive tourniquet test) and **evidence of plasma leakage**	Thrombocytopenia <100 000 cells/ mm³; HCT rise ≥20%
DHF	II	As in Grade **I plus** spontaneous bleeding.	Thrombocytopenia <100 000 cells/mm³; HCT rise ≥20%.
DHF*	III	As in Grade **I or II plus** circulatory failure (weak pulse, narrow pulse pressure (≤20 mmHg), hypotension, restlessness).	Thrombocytopenia <100 000 cells/mm³; HCT rise ≥20%.
DHF*	IV	As in Grade **III** plus profound shock with undetectable BP and pulse	Thrombocytopenia < 100 000 cells/mm³; HCT rise ≥20%.

*DHF GRADE III AND IV ARE DSS

As can be seen from the WHO table,low platelet count is currently used as a criterion for the diagnosis of DHF .

2.HIV:

Thrombocytopenia is a common finding in patients who are infected with the human immunodeficiency virus (HIV). Almost one fourth of children with HIV infection exhibit thrombocytopenia within 10 years after seroconversion[35]. In these patients both platelet destruction and impaired production are the causes of the decrease in platelet count.

Impaired production may be a direct effect of the virus, adverse effects of drug therapy, or due to associated malignancy. Regardless of the degree of thrombocytopenia, kinetic studies have shown that patients with HIV have a moderate reduction in platelet survival, but all have decreased platelet production[36]. Treatment with zidovudine has been shown to increase platelet production in some patients with HIV[37].

3.OTHERS:

Cytomegalovirus, Ebstein barr virus, Parvovirus and several other hemorrhagic viruses are associated with thrombocytopenia but the occurrence of these diseases are rare.Marrow suppression induced by the virus may be the cause of thrombocytopenia.Chronic active hepatitis may induce thrombocytopenia by causing hypersplenism which has been reported in literature.

B.BACTERIAL:

Septicemia,miliary tuberculosis, leptospirosis,typhoid and mycoplasma pneumonia are all known to cause thrombocytopenia[38].

I.SEPTICEMIA:

Thrombocytopenia is a well recognized complication seen in both gram positive as well as gram negative sepsis.Increased platelet destruction is characteristically seen in Meningococcal septicemia and often triggers DIVC in this clinical setting.Hemophagocytic histiocytosis is also seen in

septicemia and may contribute to the thrombocytopenia.Escherichia coli sepsis is associated with the hemolytic uremic syndrome. Platelet adherence to damaged vascular surfaces is seen in meningococcemia and other bacterial infections[33].Endotoxin, exotoxin and platelet activating factor may all damage the platelets,then resulting in increased clearance in the spleen.

II.ENTERIC FEVER:

Thrombocytopenia as a part of enteric fever has been documented by several workers[39].It is usually seen in complicated enteric fever and in multi drug resistant cases.This could be due to bone marrow suppression or due to liver cell dysfunction.Bleeding manifestations may also be associated with decrease in the platelet counts.Complications may be explained by the wide array of cytokines and other inflammatory mediators that are induced by Salmonella .

III.OTHER BACTERIAL INFECTIONS:

Leptospirosis is the classical example of PUO with high fever,renal compromise,elevated liver enzymes,jaundice and thrombocytopenia. Typhus group of infections is also associated with increased capillary permeability,thrombocytopenia,bleeding manifestations and skin lesions and is a close differential diagnosis for dengue fever.Mycoplasma and Tuberculosis may rarely have a decreased platelet count.

C.PROTOZOAL:

MALARIA:

Malaria is the classical protozoan disease very commonly associated with anemia and thrombocytopenia. Human platelets have been demonstrated to contain plasmodia species.Thrombocytopenia occurs in over 80% of patients with malaria. Experimental data suggested immune mediated destruction with elevated platelet activated immunoglobulin to be the cause. However in 1993, it was demonstrated that ultra structural changes in platelets, as well as the high level of parasitemia were the cause for thrombocytopenia. Antibodies against platelet absorbed microbial antigens are also responsible[40].

III.ITP:

Idiopathic Thrombocytopenic Purpura is the prototype of paediatric destructive thrombocytopenias.Other causes of consumption of platelets are autoimmune disorders including SLE.

ITP is the most common immune mediated thrombocytopenia in children[41]. ITP can be acute or chronic.Acute form is commoner in children and is seen in children 2-6 yrs old where platelets return to normal in 6 months[42]. The typical case of symptomatic childhood ITP is characterized by the sudden appearance of bruising or mucocutaneous bleeding in an otherwise healthy child,often after a preceding viral

illness.Association with mumps,measles and rubella has been documented.It is basically a diagnosis of exclusion and there should be no lymphadenopathy or hepatosplenomegaly. The severity of bleeding symptoms in childhood ITP is proportionate to the degree of thrombocytopenia.

Pharmacologic therapy is not generally indicated for children who have mild to moderate thrombocytopenia as they are unlikely to have serious bleeding [43].Moderate restriction of activity is all that is needed to be advised.The primary treatment options for the newly diagnosed patient in severe cases are corticosteroids,Intravenous immunoglobulin, and anti-Rho(D) immune globulin[44].There is no role for platelet transfusion as this will lead usually to a drop in the platelet counts,rather than increase,by triggering alloimmunisation.

IV.MARROW RELATED DISORDERS:

INFILTRATIVE DISORDERS:

In leukemia,especially ALL(Acute Lymphoblastic Leukemia),and also in Hodgkins lymphoma,Non Hodgkins lymphoma,Gauchers disease,Osteopetrosis,Myelofibrosis and Histiocytosis, thrombocytopenia is seen.Physical replacement of marrow by the tumor cells leading to decreased platelet production is the etiology in many cases.But inhibitory factors produced by the infiltrating cells are toxic to the cells of the

megakaryocytic lineage which may also contribute. The marrow shows decreased megakaryocytes, which may be larger than normal.This is a compensatory physiologic response to the infiltrative process. Autoimmune thrombocytopenia, DIVC, and thrombotic microangiopathy may all contribute to thrombocytopenia in malignancy.[45]

HEMOPHAGOCYTIC SYNDROME:

This is a condition quickly gaining prominence among clinicians in recent times.It is a correctable cause of hematological abnormalities if identified early.Guidelines for diagnosis have been established.The hemophagocytic syndrome is characterized by pancytopenia and morphologic evidence of phagocytosis of red cells, granulocytes, and platelets by reactive macrophages. It could be fatal if untreated.It is characterized by high fever, weight loss, prominent hepatosplenomegaly, severe pancytopenia and elevated liver enzymes[46].Viral etiology has been proposed.Severe, but potentially reversible, hemophagocytosis can be seen in patients with certain unusual infections (e.g., Babesiosis, Ehrilichiosis). Hemophagocytosis has also been observed in patients with SLE, Still's disease, and HIV infection[47]. Treatment should be directed to the underlying illness, with steroids and blood product support as and when needed.

HYPERSPLENISM:

Hypersplenism is a syndrome characterized by splenomegaly and any or all of the following cytopenias: anemia, leucopenia, or thrombocytopenia.The cytopenias will be corrected following splenectomy[48].Splenomegaly is almost always present in hypersplenism. Thalassemia is the most common paediatric hemolytic disorder that can present with hypersplenism.Increased splenic platelet pooling is the cause of thrombocytyopenia in hypersplenism[49]. A massively enlarged spleen can hold > 90% of the total platelet mass. In hypersplenism, the platelet count is usually between 50-150×10^9/L, and almost never <20×10^9/L. Therefore, patients with hypersplenism almost never have evidence of bleeding attributable to thrombocytopenia, nor do they need specific interventions to raise the platelet count.

OTHER MARROW CAUSES:

Aplastic anemia involves multiple hematopoietic lineages, although isolated thrombocytopenia can be the presenting feature[50].

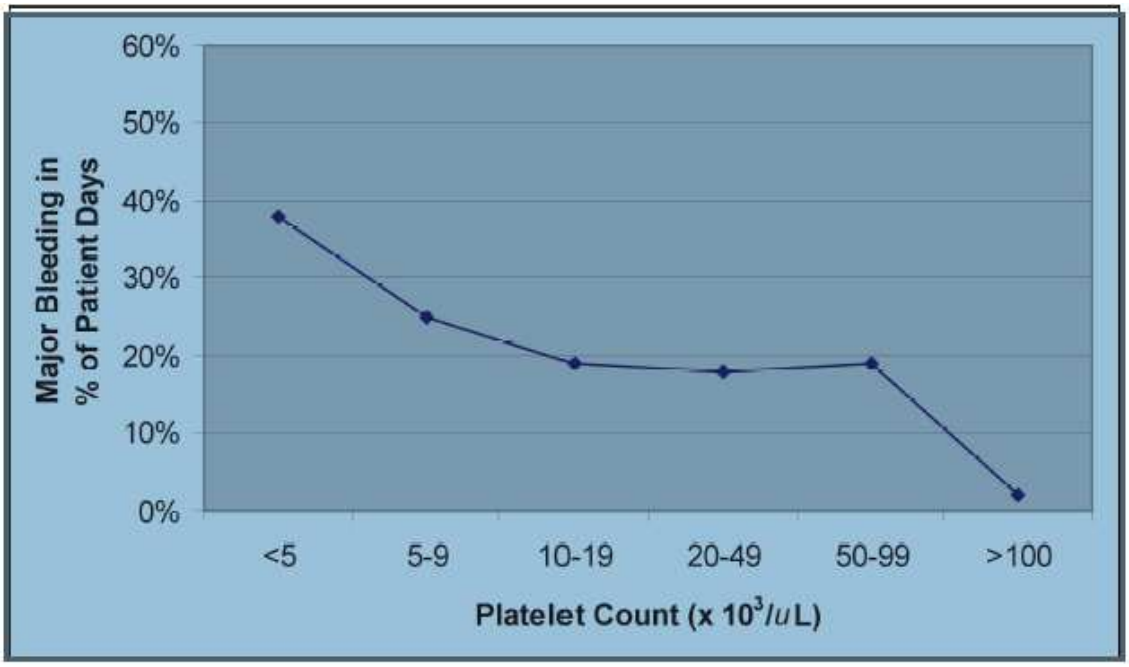
There are also numerous congenital disorders and syndromes that can be associated with thrombocytopenia.They are rare in clinical settings.Some of these conditions are Wiskott Aldrich syndrome variants and other X-linked recessive thrombocytopenias, Congenital

amegakaryocytic thrombocytopenia, Thrombocytopenia with absent radius syndrome and May Heggelin anomaly.[51]

V.OTHERS:

There are several other conditions which also can have thrombocytopenia.These include hemorrhagic snake bite,drug induced thrombocytopenia(including heparin induced thrombocytopenia),severe iron deficiency anemia,megaloblastic anemia,heart diseases,artificial valves,Kasabach Merritt syndrome,decompensated liver disease with portal hypertension and Von Willebrands disease.

FIGURE 4:CORRELATION BETWEEN THE PLATELET COUNT AND THE RISK OF BLEEDING:



Correlation between the platelet counts and bleeding risk shows increased bleeding with lower counts**.**

RELATIONSHIP BETWEEN PLATELET COUNT AND BLEEDING RISK[52]

In several clinical conditions,there is poor correlation between the counts and the incidence of bleeding.But in general,the risk of bleeding does not increase until the platelet count falls significantly below 1 lakh/L.A platelet count greater than 50000/L is adequate for hemostasis in most circumstances and most of these patients are only incidentally diagnosed.Patients who have moderate thrombocytopenia between 30 and

50000/L may only develop trivial bruising or bleeding,even with significant trauma.

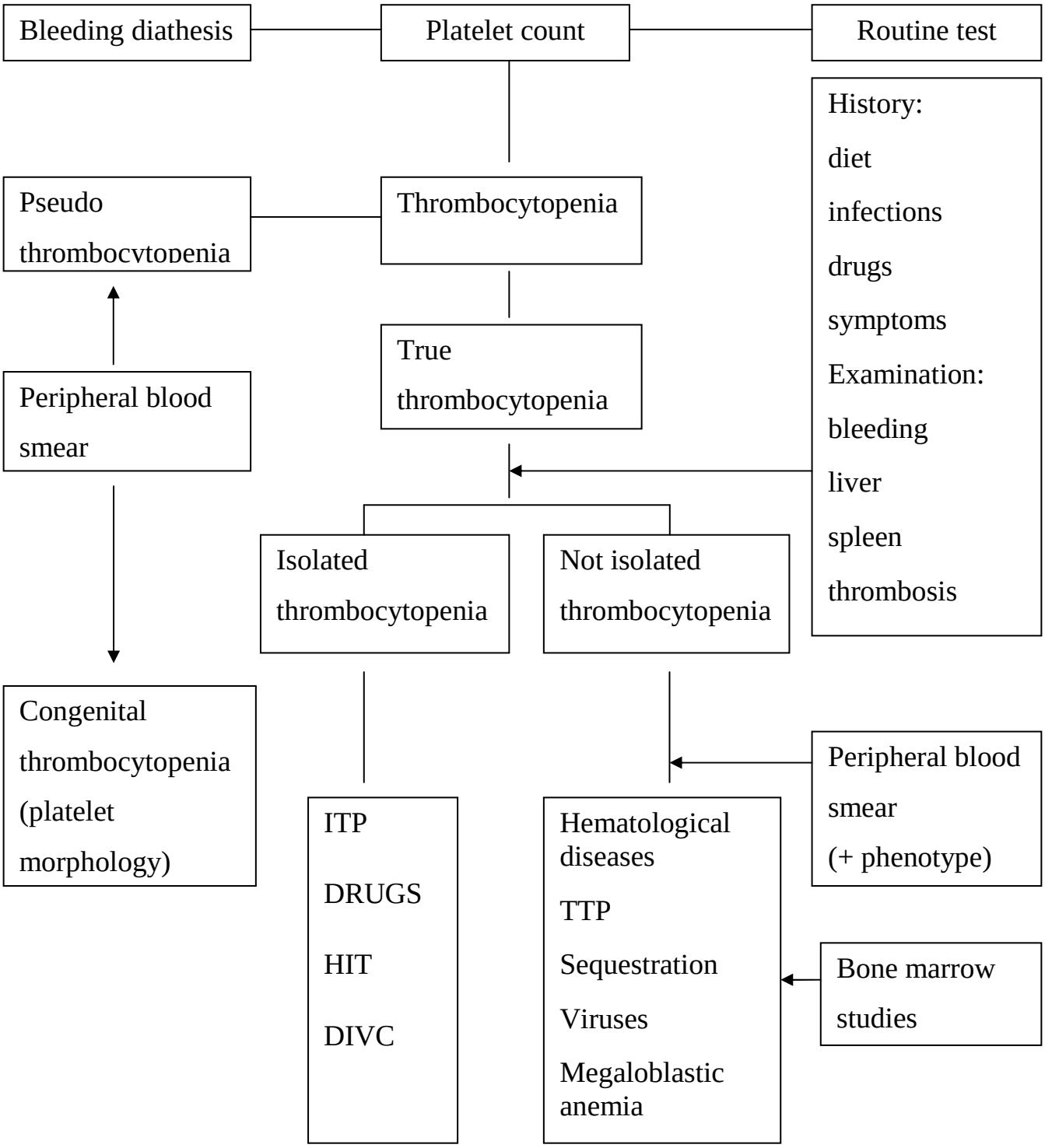
In patients with platelet counts between 10000-30000/L,there may be risk for excessive bleeding only with significant trauma. Spontaneous bleeding typically does not occur unless the platelet counts are less than 10000/L.Petechiae and spontaneous bruising may be seen in these patients,but even they may be entirely asymptomatic.It appears that the platelet count must be less than 5000 to cause critical spontaneous bleeding (eg,atraumatic intracranial hemorrhage[ICH]).

Patients who have destructive thrombocytopenias have less severe bleeding symptoms than patients who have a similar degree of thrombocytopenia due to impaired platelet production.This is because of the brisk production of younger platelets in destructive thrombocytopenias,and thus less severe bleeding symptoms.Comparatively,in conditions where there is impaired production of platelets in the marrow,the circulating population of platelets is older and ineffective[47].Hence,the bleeding manifestations seen in these conditions are also much more severe.

INVESTIGATIONS:

The figure shown below is a useful diagnostic workup plan for thrombocytopenia.

FIGURE 5: WORKING DIAGNOSTIC ALGORITHM FOR THROMBOCYTOPENIA:[53]



In evaluating a patient with thrombocytopenia, a key step is to first rule out "pseudo-thrombocytopenia," particularly in a patient without an apparent cause for the thrombocytopenia. Once pseudothrombocytopenia is ruled out,if a low platelet count is obtained in EDTA-anticoagulated blood, a blood smear should be evaluated and a platelet count determined in blood collected into sodium citrate or heparin tube, or a smear of freshly obtained anticoagulated blood, such as from a finger prick, can be examined.

The diagnostic work up of patients with thrombocytopenia should then include the following battery of investigations:

1. Complete Blood Count: Presence of other cytopenias should be looked for.Mean platelet volume needs to be assessed. A mildly elevated MPV is consistent with a destructive cause.

A. Total count, Differential Count

Leucopenia-in early dengue before IgM ELISA is positive Leukocytosis-predominant neutrophils indicate bacterial infection, septicemia, predominant lymphocytosis in Tuberculosis.

2. E.S.R: elevated in most of the infections, malignancies, anemia .It is a non specific test.

3. Peripheral blood smear[54]: The ability to assess the Peripheral blood smear accurately is invaluable. Malarial parasites and gametocytes, Dohle bodies, thrombocytopenia, abnormal cell morphology in leukemia should

be carefully looked for. The smear should be examined to estimate the platelet number (1 platelet/high power field=platelet count of 10-15000).Platelet clumps,variation in the size of platelets,evidence of hemolysis in the form of spherocytes and schistocytes are to be looked for.Direct coombs test will be needed if the smear is suggestive of hemolysis.Presence of malarial parasites and ring forms in the RBC's are important in endemic areas.

4. Blood sugar, urea, creatinine -acute renal injury may be caused by infections like malaria and leptospirosis,septic shock,DSS.

5. Liver function tests- hyperbilirubinemia and elevated liver enzymes are seen in malaria (esp. falciparum), leptospirosis,septicemia,dengue.

6. Urine albumin, deposits-renal failure (malaria, leptospirosis), Urinary tract infections

7. Chest X Ray- pleural effusion in dengue, pulmonary tuberculosis

8.Ultrasonography-hepatomegaly,splenomegaly.Gallbladder pericholecystic edema with wall thickening is suggestive of dengue.Ascites can also be there in dengue.

9. Widal- serological method for evaluation of typhoid fever.Should be done in the second week of fever.

10.dengue panel-NS1 is positive from days 2-9 of the illness.IgM Dengue ELISA is positive after 5^{th} day of fever and rising titres are indicative of

dengue.Similarly IgG Dengue ELISA-four fold increase in titre is highly suggestive of dengue.

TABLE 4: INTERPRETATION OF DENGUE DIAGNOSTIC TESTS:[55]

Highly suggestive	Confirmed
One of the following: 1. IgM + in a single serum sample 2. IgG + in a single serum sample with a HI titre of 1280 or greater	One of the following: 1. PCR + 2. Virus culture + 3. IgM seroconversion in paired sera 4. IgG seroconversion in paired sera or fourfold IgG titer increase in paired sera

11. IgM ELISA leptospiral antibodies-Acute toxic presentation with conjunctival suffusion, renal failure and abnormal liver function tests

12. Blood culture – at least 3 blood culture samples to be taken, special technique is required for fastidious organisms to grow.

13. Urine culture and sensitivity-urinary tract infections

14. Bone marrow examination: Leukemia, lymphoma, Pyrexia of unknown origin,pancytopenia.In patients with fever and thrombocytopenia with renal and liver parameters being abnormal, it is very important to consider a bone marrow biopsy which may help to differentiate inadequate production from excessive destruction/consumption as the predominant cause of thrombocytopenia [56]. A bone marrow examination is not necessary in most cases of isolated unexplained thrombocytopenia in children.In general, a bone marrow examination is indicated for pancytopenia when

there are blasts in peripheral smear,and systemic symptoms such as fever, fatigue, weight loss,or bone pain.

ROLE OF PLATELET TRANSFUSIONS IN THROMBOCYTOPENIA

In 1910, W.W Duke,a JOHNS HOPKINS physician demonstrated the relation of blood platelets to bleeding and demonstrated for the first time that platelet transfusion could have a role in relieving it[57]. But until 1950,reliable methods for platelet preparation were not developed.Even in 1970,only specialized medical centers had facilities for platelet transfusion.However,today,platelets are easily available on an emergency basis.

Guidelines for platelet transfusions of children and adolescents are comparable to adult guidelines as shown in table 5.When platelet levels fall to <20000/L ,spontaneous bleeding risk increases markedly, if serious complications (infection, organ failure, clotting abnormalities, or anemia) are present. In this setting, prophylactic transfusions are given to maintain a count of >20000/L. However, in practice, severe thrombocytopenia commonly occurs in association with the complications of fever, antimicrobial therapy, active bleeding, disseminated intravascular coagulation, and other severe clotting abnormalities, situations in which

transfusions are given to maintain relatively high counts.Hence guidelines are arbitrary and have to be judiciously applied in the clinical setting.

Long term and repeated transfusions in any patient may lead to alloimmunization and refractoriness. The goal of most platelet transfusions is to raise the platelet count to >50000/L for older children and >100000/L for neonates. This can be achieved consistently in children weighing up to 30 kg by infusing 10 mL/kg of standard (unmodified) Platelet concentrates, obtained either from processing whole blood units or by platelet pheresis. For children weighing more than 30 kg, the appropriate dose is 3–6 pooled whole blood–derived units or 1 apheresis unit. Platelet concentrates should be transfused as rapidly as the patient's overall condition permits, certainly within 2 hr. Patients requiring repeated platelet transfusions should receive leukocyte-reduced blood products, including platelet concentrates, to diminish alloimmunization and platelet refractoriness and reduce the risk of transfusion-transmitted cytomegalovirus infection.

Transfusion of 10 mL/kg of an unmodified platelet concentrate is adequate because it adds to increase the platelet count by 10000/L. Moreover, 10 mL/kg is not an excessive transfusion volume, provided the intake of other IV fluids is monitored and adjusted.However in dengue,even this volume can contribute to fluid overload with no proven benefit to patient outcome.Hence routine platelet transfusions are not

recommended in Dengue by WHO. It may be considered only in very severe thrombocytopenia less than 10,000/L.[58]

TABLE 5 -- GUIDELINES FOR PAEDIATRIC PLATELET TRANSFUSIONS[59]

CHILDREN AND ADOLESCENTS
Platelets < 50×10^9/L and bleeding < 50×10^9/L and an invasive procedure < 20×10^9/L and marrow failure with hemorrhagic risk factors < 10×10^9/L and marrow failure without hemorrhagic risk factors At any count, but with Platelet dysfunction plus bleeding or an invasive procedure
INFANTS WITHIN THE FIRST 4 MONTHS OF LIFE
Platelets< 100×10^9/L and bleeding < 50×10^9/L and an invasive procedure < 20×10^9/L and clinically stable < 100×10^9/L and clinically unstable At any count, but with Platelet dysfunction plus bleeding or an invasive procedure

In most infectious diseases, the thrombocytopenia is often transient and seldom requires platelet transfusions. Treating the underlying condition will result in drastic improvement of platelet count and its complications.

STUDIES ON THROMBOCYTOPENIA:

- A prospective study was conducted in Hayat Shaheed hospital,Peshawar by Ali jan et al,from June 1995 to December 1996.100 children with thrombocytopenia were studied.The differential diagnosis and the bleeding manifestations were documented.ITP was found to be the leading diagnosis followed by aplastic anemia and leukemia.There was good response to steroids in the study group[60].
- Another prospective study in Shri Ganga Ram hospital,Delhi by Sachdev et al was done to evaluate the outcome of children with thrombocytopenia in the paediatric intensive care unit.138 patients were included over a 7 month study period. Patients requiring cardiopulmonary resuscitation or with circulatory shock, coagulopathy, sepsis and with more severe disease were found to have higher risk of developing thrombocytopenia. Drop in platelet counts >27% and thrombocytopenia were found to be independently related to mortality[61].
- A study done in University college of Medical Sciences,Delhi by Mittal et al was done during the 2010 Dengue epidemic to assess the clinico hematological profile and platelet trends in the admitted children.It was a retrospective study which assessed 135 children.The study concluded that complications and mortality were low.There was also higher age of

presentation of dengue cases compared to previous studies and more DHF cases indicating an increase in the severity of disease.The platelet recovery time was found to be not influenced by the disease category[62].

- A study done in St Louis University,USA assessed the implications of thrombocytopenia in the paediatric ICU By Krishnan et al[63].They found an incidence of 17.3% of thrombocytopenia among PICU patients and that a 10% drop in platelet count in these patients was associated with increase in the hospital stay and mortality.

MATERIALS AND METHODS

4. MATERIALS AND METHODS:

STUDY DESIGN:

Descriptive,Cross sectional study.

STUDY POPULATION:

This study was done on children who were admitted to the children medical ward of Tirunelveli Medical College Hospital during the period from December 2011 to April 2012. 112 consecutive patients who satisfied the following inclusion criteria were studied.Prior ethical committee approval was obtained for the study.The total number of admissions during the study period was 702.

METHODOLOGY:

INCLUSION CRITERIA :

1. The patients of both sexes aged 2 MONTHS TO 12 YEARS.
2. Patients with platelet counts less than 1 lakh anytime during the course of hospital stay,irrespective of the cause for admission.

EXCLUSION CRITERIA :

1. Patients with spurious thrombocytopenia-lab induced errors where immediate repeat platelet counts or the peripheral smear did not grossly correlate with the first count were excluded

2. Patients who were earlier diagnosed to have conditions that are known to cause thrombocytopenia (e.g. known cases of hematological malignancies, aplastic anemia, MDS, ITP).
3. Patients who have already received platelet transfusion prior to admission.
4. Patients who were very sick at admission or expired within few hours of admission,who could not be subjected to the full set of investigations.

STUDY PROTOCOL:

Once the patients were included in the study,data regarding the patient was entered into preset proforma as regards to the history,general and systemic examination,Hess test and vital signs . The bleeding manifestations patients presented with or developed during their course in hospital were recorded.An awareness questionnaire on dengue with three simple questions was also included for the parents.Informed consent was obtained.

Following investigations were done for all patients as a 1ST PANEL:

1. CBC including TC,HB,ESR,PCV.The trend of platelet counts was monitored 12-24 hourly till a count of 1 lakh was reached,with or without intervention.
2. PERIPHERAL SMEAR FOR MP/MF AND FOR THE BLOOD PICTURE

3. Blood sugar, urea, creatinine,serum bilirubin,liver enzymes
4. IgM DENGUE ELISA,NS1 ELISA AND IgG DENGUE ELISA-these were done twice;1st sample on the day of onset of thrombocytopenia and the second one as a convalescent sample to identify positivity and to quantitate the rise in titre.
5. BLOOD WIDAL,URINE C/S,BLOOD C/S.
6. CHEST X RAY AND USG ABDOMEN

If the 1st panel did not reveal a diagnosis,a 2nd panel of investigations were done:

1. IgM ELISA FOR LEPTOSPIROSIS,
2. BONE MARROW STUDY
3. Antinuclear antibodies,PT,aPTT,hepatitis screening,CRP were done depending on the clinical scenario.

Once the specific diagnosis was reached, patients were treated for it specifically and symptomatically (Mechanical ventilation, shock correction,steroids).Blood products were transfused as per the treating physician's discretion. The proportion of study patients requiring interventions to improve platelet count like platelet transfusion, steroids and the reason for such interventions were recorded.

The questions in the awareness questionnaire are:

1. Have you heard the word "dengue"?

2. Tell the signs and symptoms of the disease
3. How is the disease transmitted?

The answers given by the parents were also documented.

The gathered data was fed into a master chart(annexure) to aid statistical analysis.

The causes of fever with thrombocytopenia are so numerous, a simple workable classification is presented –

1. Viral causes : Dengue; Parvo-B19; hepatitis, HIV,CMV
2. Bacterial causes : Gram positive and negative septicemia, miliary tuberculosis,leptospirosis, typhoid etc.
3. Protozoal causes : Malaria.
4. ITP/TTP/HUS
5. Others : Leukemia, lymphoma,hypersplenism,aplastic anemia etc..
6. DIVC-sepsis,snake bite.
7. Connective tissue disorders-SLE.

COLLABORATING DEPARTMENTS:

Departments of Biochemistry,Microbiology and Pathology; Tirunelveli Medical College, Tirunelveli.

LIMITATIONS OF THE STUDY:

1. Study was done during the seasonal period for infectious diseases.Hence incidence of infectious diseases might have been

higher

2. Complete bleeding profile in the form of PT,aPTT were not done for all the patients.
3. Incidence of malaria is very low as the region is not an endemic area.Hence the data on malaria is subject to confounding.
4. Drug induced thrombocytopenia was not studied.
5. Viral serology was not done due to financial constraints.

STATISTICAL ANALYSIS :

Data was entered into an Excel Spreadsheet and analysed using SPSS Version 16. Using this software, frequencies, percentages, means, standard deviations, chi square test, paired t test, unpaired t test correlation were applied. A 'p' value less than 0.05 is considered significant.

OBSERVATION & RESULTS

5. OBSERVATIONS AND RESULTS

Total number of admissions during the study period in children medical ward is 702.Number of patients who had thrombocytopenia or developed it subsequently during the course of hospital stay is 112(after application of the exclusion criteria),which means one among every 6.25 children admitted developed thrombocytopenia.(15.95%incidence)

In 107 patients a cause for the thrombocytopenia could be identified with the panel of investigations applied.5 patients were left undiagnosed despite full battery of investigations.

TABLE 6: AGE WISE DISTRIBUTION AND MORTALITY

S.NO	AGE	Frequency (n=112)	Percent	Mortality	Percent	p value
1	<1 year	11	9.8	5	45.5	0.073
2	1-5yrs	33	29.5	1	3	
3	6-10yrs	53	47.3	2	3.8	
4	>10 yrs	15	13.4	0	0	

The commonest age group of presentation of thrombocytopenia among the study group is 6-10 years,constituting 47.3% of the cases.Mortality is highest among infants.Out of 11 infants studied,5 expired(45.5%).But this is not statistically significant($p>0.05$).Mean age of

presentation is 6.56 years(SD=3.49).The mean age of expired children is 2.8years which is statistically significant(**p<0.05**).

TABLE 7: SEX WISE DISTRIBUTION OF THROMBOCYTOPENIA

S.NO	SEX	NO OF PATIENTS (N=112)	PERCENTAGE
1	MALE	53	47.3
2	FEMALE	59	52.7

52.7% of the study population were girl children.There is no particular sex predilection for thrombocytopenia.The Male female ratio is 0.89:1.

TABLE 8: OUTCOME OF CHILDREN WITH THROMBOCYTOPENIA

S.NO	OUTCOME	N=112	PERCENTAGE
1	DISCHARGE	97	86.6
2	DEATH	8	7.1
3	REFERRED	7	6.3

8 patients expired during the course of the hospital stay.The mortality rate is 7.1%.For 7 patients there was no significant improvement in platelet count till discharge because of the underlying disease process as in leukemia,hypersplenism and ITP and were referred to higher institutions.In all the other patients(86.6%) the platelet count improved prior to discharge and the thrombocytopenia was only transient.

FIGURE 6:COMPARISON OF AGE WISE INCIDENCE AND THE MORTALITY IN EACH AGE GROUP

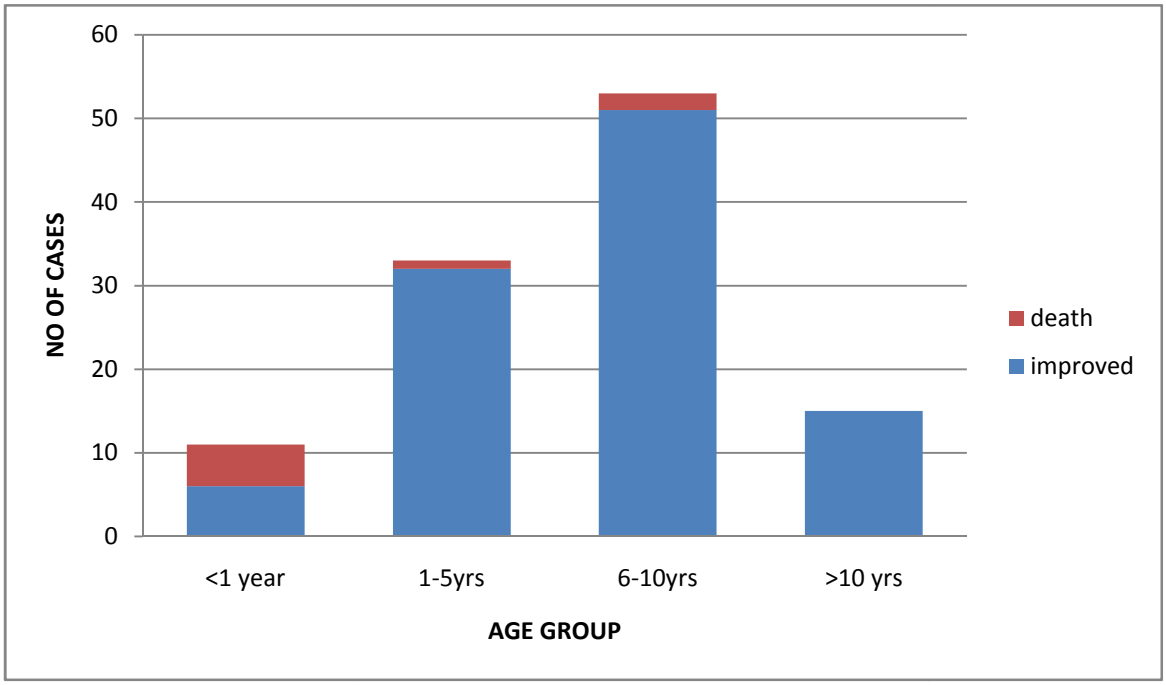


FIGURE 7: SEX WISE DISTRIBUTION

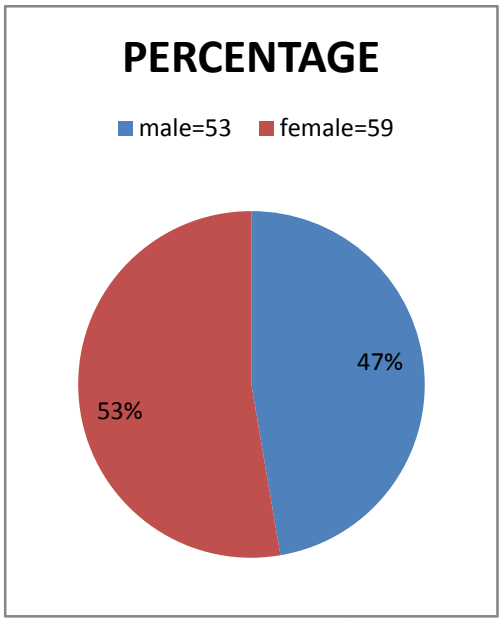


FIGURE 8:OUTCOME

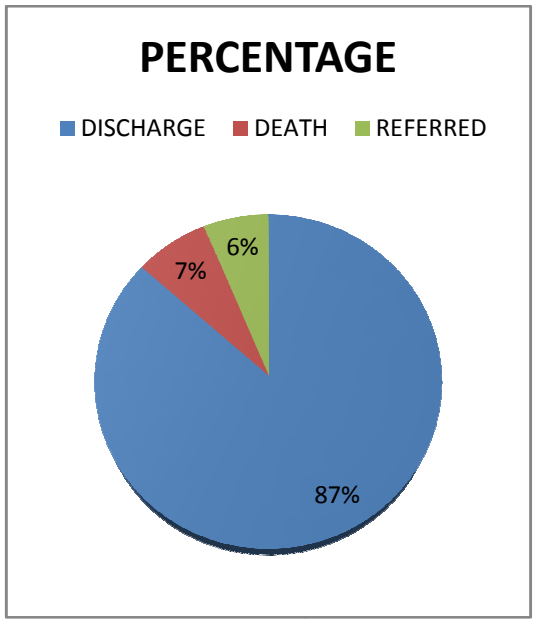


TABLE 9: ETIOLOGY AND DISEASE WISE MORTALITY OF THROMBOCYTOPENIA

s.no	DIAGNOSIS	Frequency(n=112)	Percent	Death(n=8)	%
1	Dengue fever(DF)	36	32.1	0	0
2	DHF	21	18.7	0	0
3	DSS	9	8	4	44.4
4	Enteric	13	11.6	0	0
5	Dengue/enteric co infection	4	3.6	0	0
6	Malaria	3	2.7	0	0
7	ALL	5	4.5	0	0
8	Septicemia	5	4.5	2	40
9	Undiagnosed	5	4.5	0	0
10	Miscellaneous	11	9.8	2	18.2

The commonest etiology for newly diagnosed thrombocytopenia among children admitted is Dengue.Total dengue cases were 66,comprising 58.8% of the study population.Among the dengue cases,dengue fever with or without hemorrhage(DF) was most common(32.1%).The second commonest diagnosis was enteric fever.11.6% of the thrombocytopenia cases had enteric fever.In 5 cases,a final diagnosis could not be reached(4.5%),which is within allowable limits.All 3 cases of malaria were due to Plasmodium vivax.All cases of leukemia were acute lymbhoblastic leukemia(ALL).4 cases were due to co infection with both dengue and enteric fever,one of which also had urinary tract infection.

Leading cause of mortality in the study population is dengue shock syndrome(DSS),causing 4 out of the 8 total deaths.DSS comprised only 8% of cases with thrombocytopenia,but had the highest mortality rate of 44.4%.The next leading cause of mortality was septicemia.

TABLE 10:MISCELLANEOUS CAUSES OF THROMBOCYTOPENIA

S.NO	DISEASE	FREQUENCY (n=11)
1	ITP	2
2	Snake bite	2
3	Leptospirosis	1
4	Hepatitis	1
5	HIV	1
6	Lymphoma	1
7	Aplastic anemia	1
8	Hemophagocytic syndrome	1
9	Hypersplenism	1

11 cases had rarer diagnosis.2 of these patients died.Cause of death in one case was snake bite with severe bleeding manifestations probably DIVC.The other death was a diagnosed case of hemophagocytic syndrome with elevated ferritin and triglycerides and a marrow picture showing presence of histiocytes.

FIGURE 9: CHART SHOWING THE ETIOLOGY AND DISEASE WISE MORTALITY

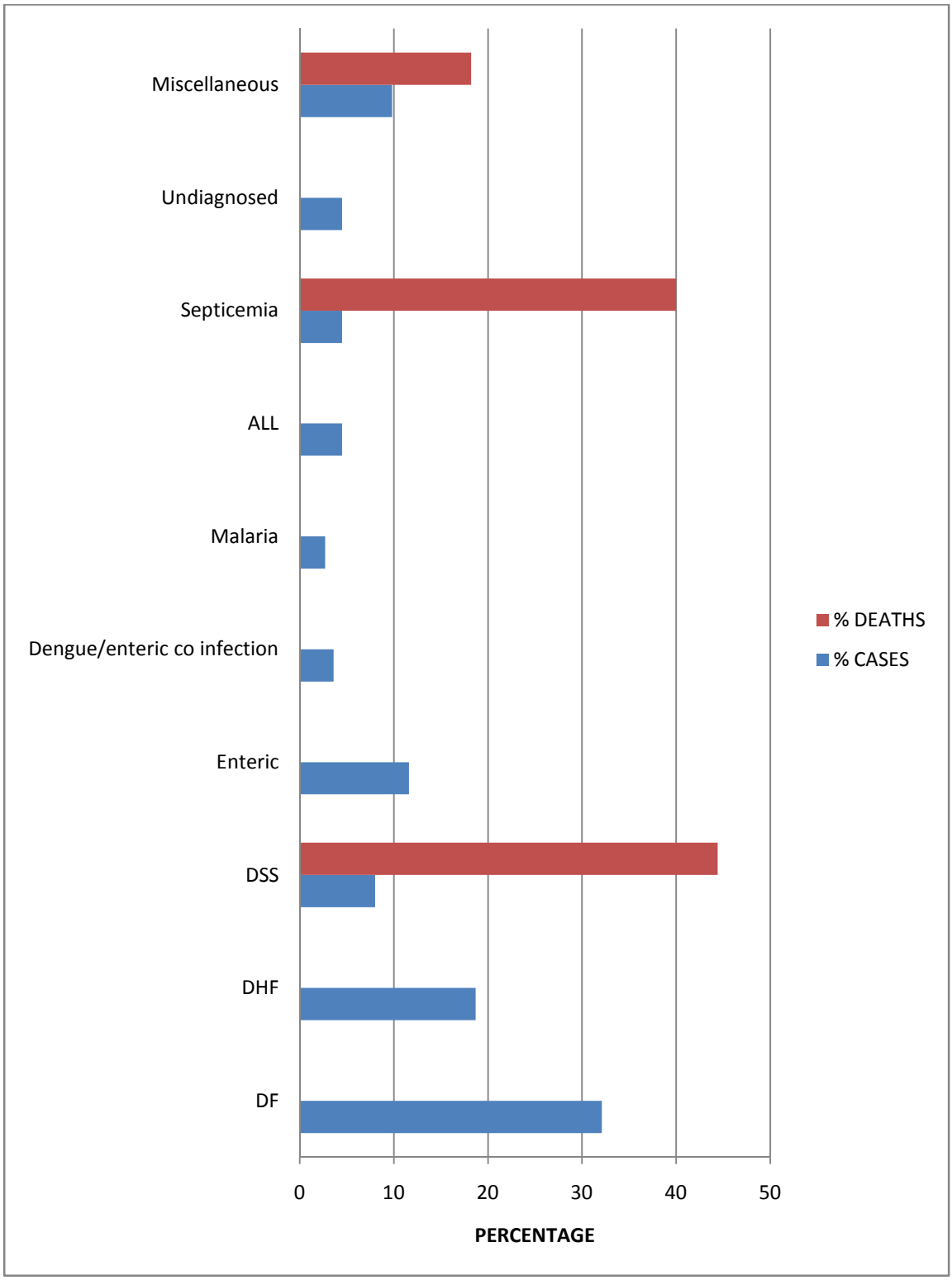


TABLE 11: ETIOLOGY IN 107 DIAGNOSED CASES

DIAGNOSIS	FREQUENCY	PERCENTAGE
INFECTIVE	94	87.9
NONINFECTIVE	13	12.1
TOTAL	107	100

Infections caused most of the thrombocytopenia.Commonest non infective cause was ALL.

TABLE 12:COMPARISON OF PREHOSPITAL TREATMENT RECEIVED AND OUTCOME

s.no.	Pre hosp	No. of cases(n=84)	Deaths n=8(%)	Adequacy of treatment received(%)
1	Government hospitals	34(40.48)	1(2.9)	76.5
2	Phc's	3(3.57)	0	33.3
3	Private hospitals	47(55.95)	5(10.6)	68.1

Among the study group,75% were referred cases.6 of the total 8 deaths were among referred cases.The highest number of referrals were from private institutions(55.95%),10.6% of whom expired.The adequacy of treatment received was assessed by the proper administration of intravenous fluids,antibiotics, blood products and early referral.District headquarters government hospitals have adequately treated and referred patients(76.5%).

TABLE 13: FEVER DURATION AT ADMISSION AND OUTCOME

S.NO.	fever in days on adm	Frequency n=112	Percentage	Deaths n=8	%deaths
1	1-4 days	41	36.6	3	7.3
2	5-7days	50	44.6	3	6
3	8-10 days	10	8.9	1	10
4	>10 days	6	5.4	0	0
5	afebrile	5	4.5	1	20

p=0.169

44.6% of the children presented with fever of 5-7 days duration.5 cases had no fever at admission.There is no correlation between early presentation and the outcome($p>0.05$).Mean duration of fever at admission is 5.69 days.(SD=3.03)

FIGURE 10:COMPARISON OF OUTCOME AMONG REFERRAL CASES

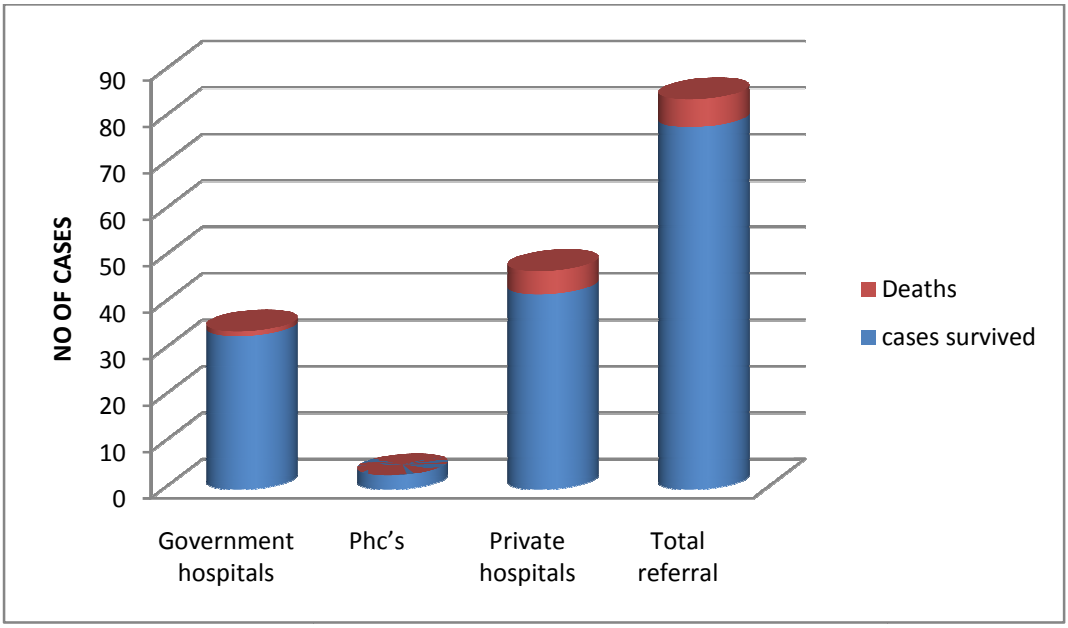


FIGURE 11: COMPARISON OF NUMBER OF DAYS OF FEVER AT ADMISSION AND THE MORTALITY

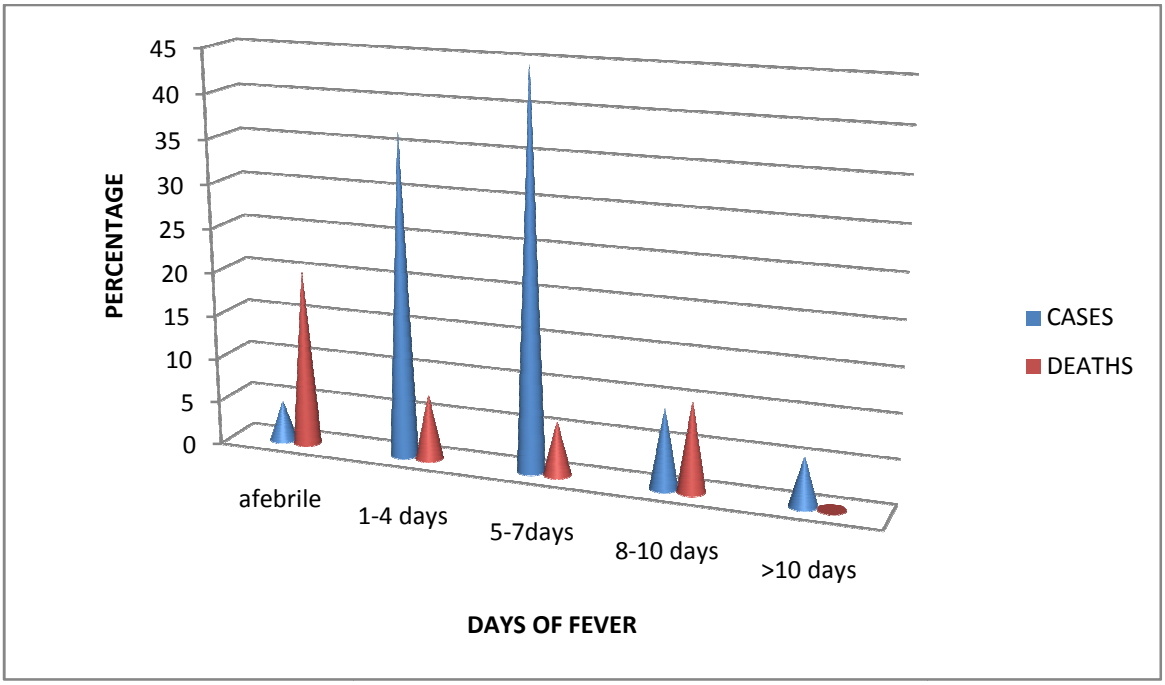
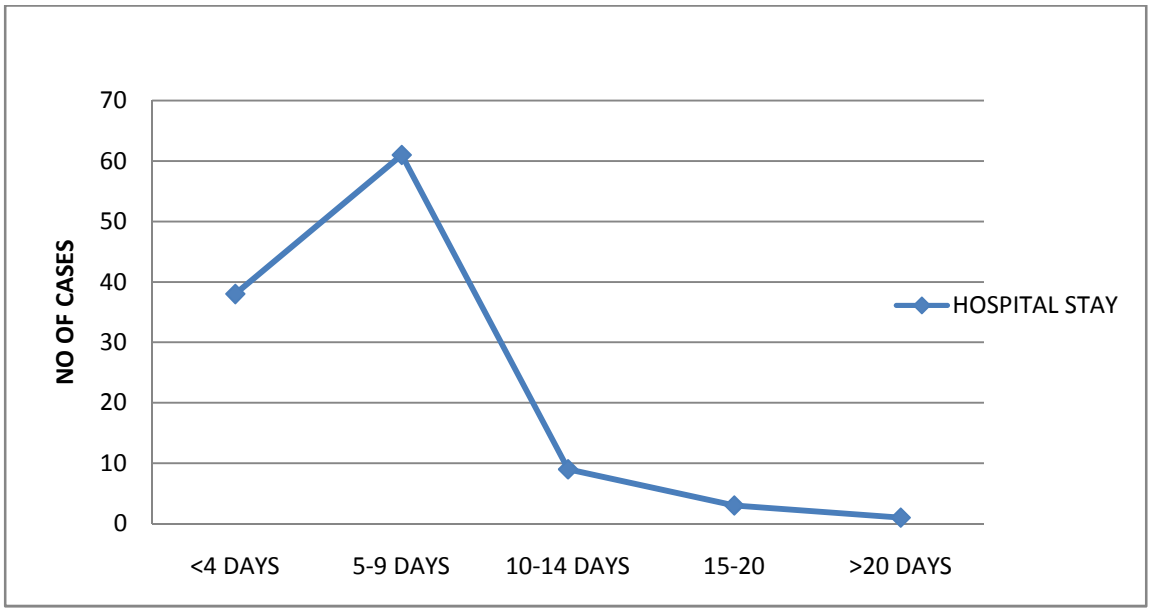


FIGURE 12: DURATION OF HOSPITALISATION AS INDICATOR OF MORBIDITY AND MORTALITY



Mean duration of hospital stay in the study group is 6.15 days.Children who expired had a significantly**(p<0.05)** short hospital stay(2.38 days) compared to survivors(6.44 days),indicating they were probably very sick at admission.

TABLE 14: TOTAL DURATION OF FEVER AND OUTCOME

S.NO.	Total days of fever	FREQUENCY n=112	%	Death n=8	% p=0.383
1	<4 days	12	10.7	3	25
2	5-7	53	47.3	2	3.8
3	8-10	24	21.4	1	4.1
4	10-15 days	14	12.5	0	0
5	>15 days	4	3.6	1	25

Mean duration for which fever lasted is 7.69 days(SD=3.498).47.35% children became afebrile after 5-7 days. Children who had prolonged fever more than 15 days had the worst outcome(25% mortality).But this is not statistically significant(p=0.383)

TABLE 15: REPORTS OF AWARENESS STUDY

S.NO.	parameter	no of parents	%
1	heard of dengue	43	38.4
2	signs and symptoms	20	17.9
3	Knew transmission	29	25.9
4	Complete knowledge	12	10.72

Only 38.4% of the parents had ever heard of the word dengue.17.9% knew the signs and symptoms of the disease and 25.9% knew that the disease was spread by mosquitoes.Only 10.7% parents had the basic knowledge about the disease that is expected of a layman,indicating poor awareness of the disease.

FIGURE 13:COMPARISON OF TOTAL DAYS OF FEVER AND MORTALITY

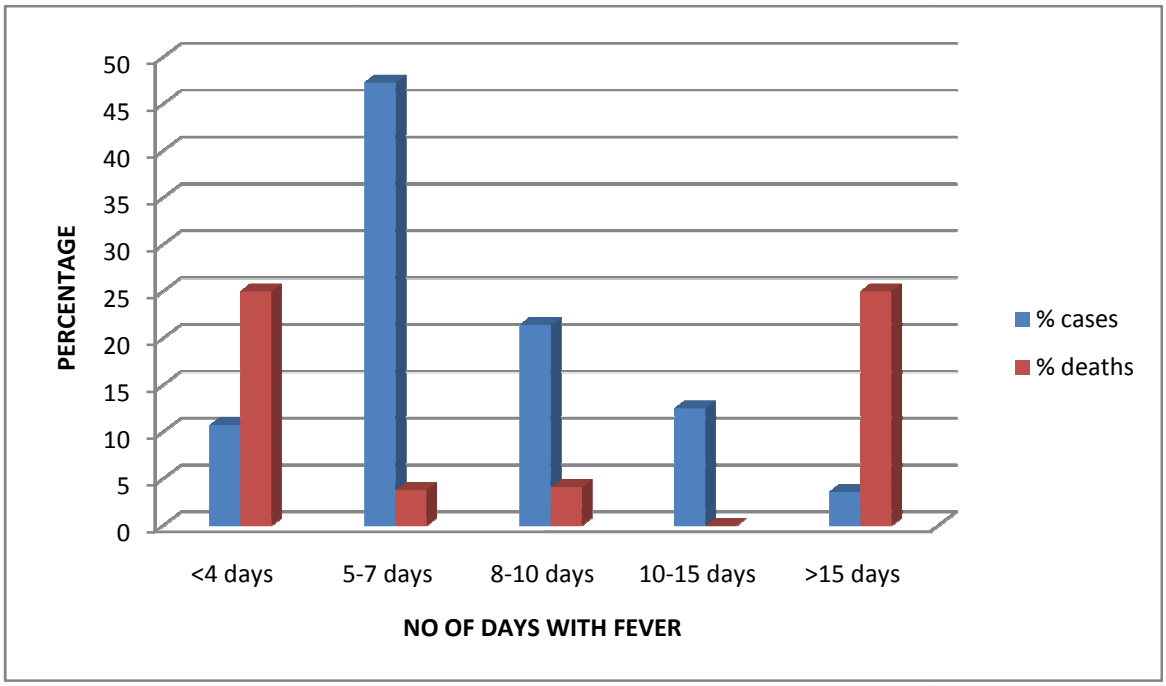


FIGURE 14:COMPARISON OF THE AWARENESS TRENDS

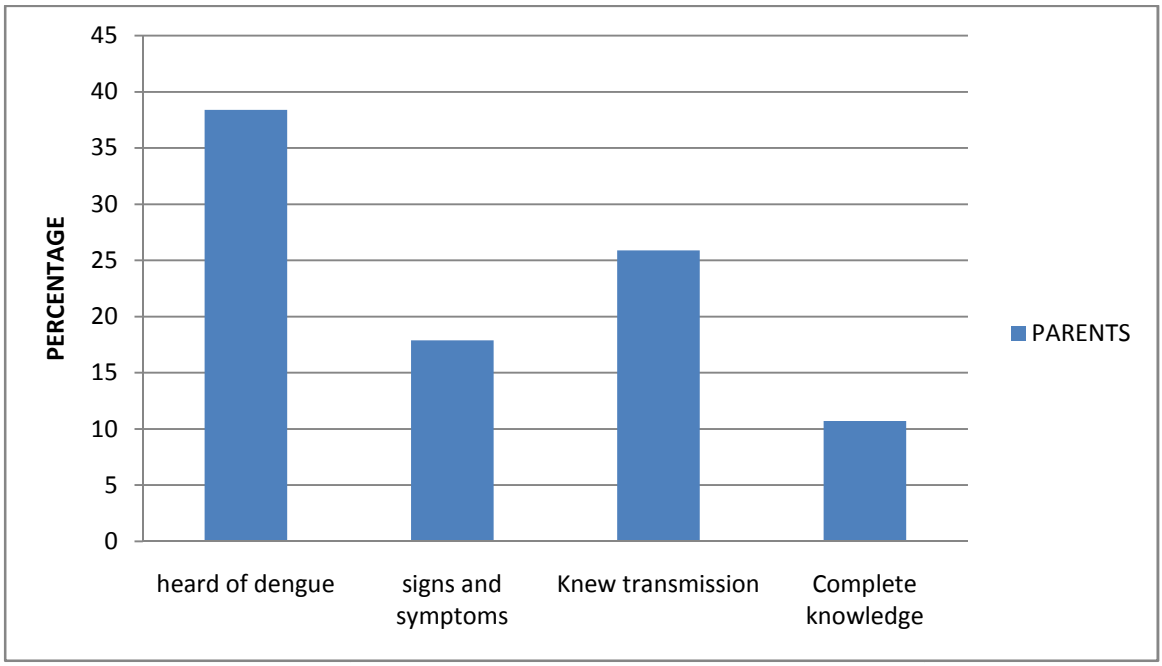


TABLE 16: SYMPTOM ANALYSIS OF CASES BASED ON ETIOLOGY

Features	total n=112(%)	dengue n=66(%)	enteric n=13(%)	D/E n=4(%)	ALL n=5(%)	sepsis n=5(%)
Fever	107(95.5)	66(100)	13(100)	4(100)	5(100)	5(100)
abd pain	59(52.7)	35(53)	9(69.2)	3(75)	1(20)	0
Vomiting	73(65.2)	42(63.6)	11(84.6)	3(75)	2(40)	4(80)
Cough	42(37.5)	23(34.9)	3(23)	3(75)	1(20)	5(100)
Myalgia	60(53.6)	35(53)	6(46.2)	2(50)	3(60)	0(0)

p>0.05

The most common presenting symptom among the study group is fever(95.5%) with vomiting being the second most common symptom(65.2%).All the major diagnosed diseases,including leukemia had fever.Abdominal pain was most common in dengue/enteric co infection while vomiting was most common among enteric fever cases. None of these symptoms are statistically significant as far as outcome is concerned(p>0.05).

76.1% of children with vomiting had bleeding manifestations.This is statistically significant(**p=0.003**).

TABLE 17:CLINICAL SIGNS IN THE VARIOUS DISEASES

S.NO.	Features	Total n=112(%)	dengue n=66(%)	enteric n=13(%)	D/E n=4(%)	sepsis n=5(%)
1	abd distension	28(25) **p =0.001**	16(24.2)	3(23)	1(25)	2(40)
2	abd tenderness	18(16.1)	12(18.2)	3(23)	1(25)	0
3	Oliguria	15(13.4)	6(9.1)	2(15.4)	1(25)	3(60)
4	Puffy face	27(24.1)	16(24.2)	2(15.4)	0	2(40)
5	pedal edema	11(9.8) **p=0.006**	6(9.1)	1(7.7)	0	0
6	Erythema/ flush	54(48.2)	38(70.4)	5(9.3)	3(5.6)	1(1.9)

Erythematous flush was present in 48.2% of the cases.70.4% of dengue cases had erythematous rash.Oliguria,facial puffiness and abdominal distension were the commonest in cases with septicemia.Enteric/dengue co infection cases had more abdominal tenderness compared to other cases.Abdominal distension and pedal edema were significantly associated with low platelet counts, bleeding manifestations,increased transfusion needs and a poor outcome**(p<0.05)**.

FIGURE 15:COMPARISON OF THE CLINICAL SYMPTOMS BETWEEN DENGUE AND ENTERIC FEVER

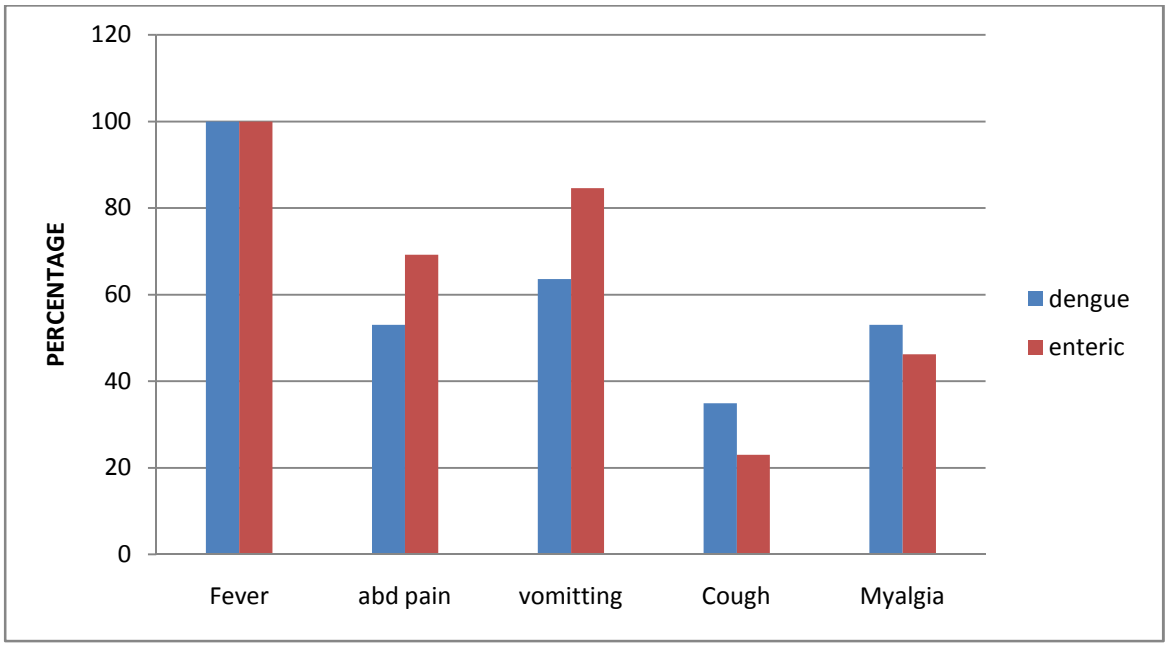


FIGURE 16:COMPARISON OF THE CLINICAL SIGNS BETWEEN DENGUE AND ENTERIC FEVER

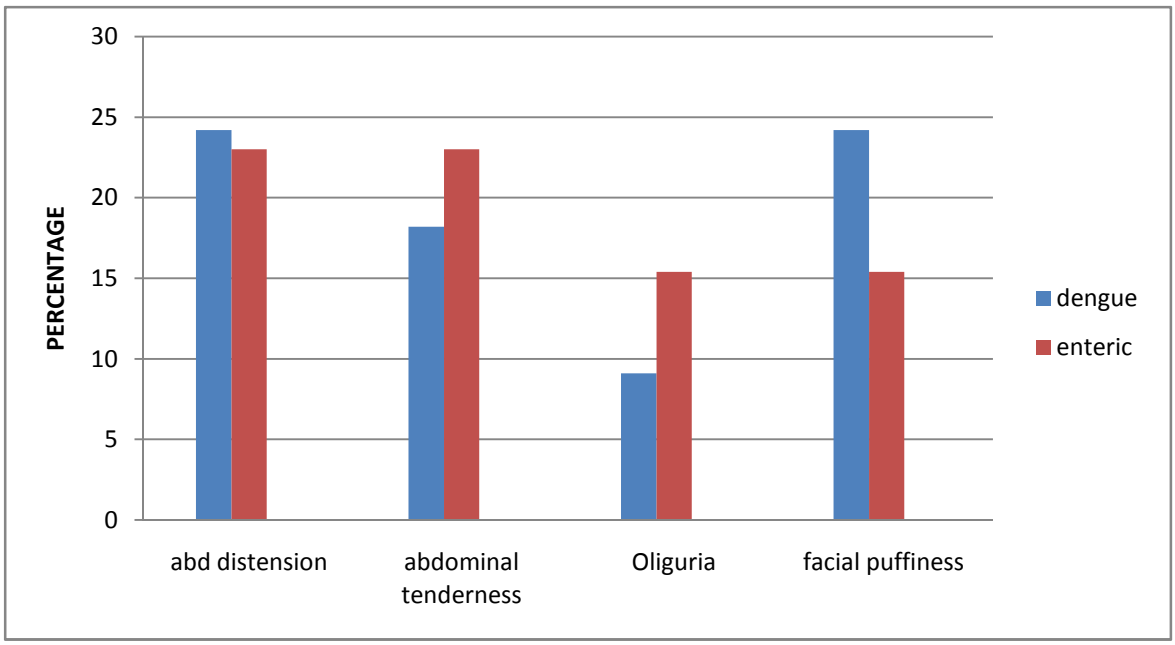


TABLE 18:PREDICTORS OF MORTALITY IN VARIOUS DISEASES

features	total n=112(% of n)	death n=8(%)	p value outcome	Dengue deaths n=4(%)	septicemia deaths n=2(%)
altered sensorium	49(43.8)	8(100)	**0.001**	4(100)	2(100)
tachycardia	48(42.9)	8(100)	**0.001**	4(100)	2(100)
tachypnea	20(17.9)	8(100)	**0.000**	4(100)	2(100)
shock	18(16.1)	8(100)	**0.000**	4(100)	2(100)
seizure	13(11.6)	3(37.5)	**0.018**	1(25)	2(100)
mech vent	7(6.3)	6(75)	**0.000**	2(50)	2(100)
inotrope	8(7.1)	7(87.5)	**0.000**	3(75)	2(100)
narrow pulse pressure<20	19(17)	2(25)	0.484	1(25)	1(50)
malnutrition	63(56.3)	1(12.5)	**0.041**	1(25)	0

In children with thrombocytopenia, the presence of Altered sensorium,tachycardia,tachypnea,shock at presentation,seizures were all significantly associated with low platelet counts,bleeding and mortality(**p<0.05**).Children requiring inotrope support,mechanical ventilation also had poor outcome(**p<0.05**).The mortality was also significantly high(**p<0.05**) in malnourished children with thrombocytopenia .In fact ,the mean weight of the expired children was only 10kg compared to 17kg in survivors.Hence,all these factors are to be considered significant predictors of mortality. Narrow pulse pressure has not significantly affected the outcome.Occurence of seizures in cases with septicemia and thrombocytopenia had strong correlation with death(100%) .

FIGURE 18:COMPARISON OF PREDICTORS OF MORTALITY

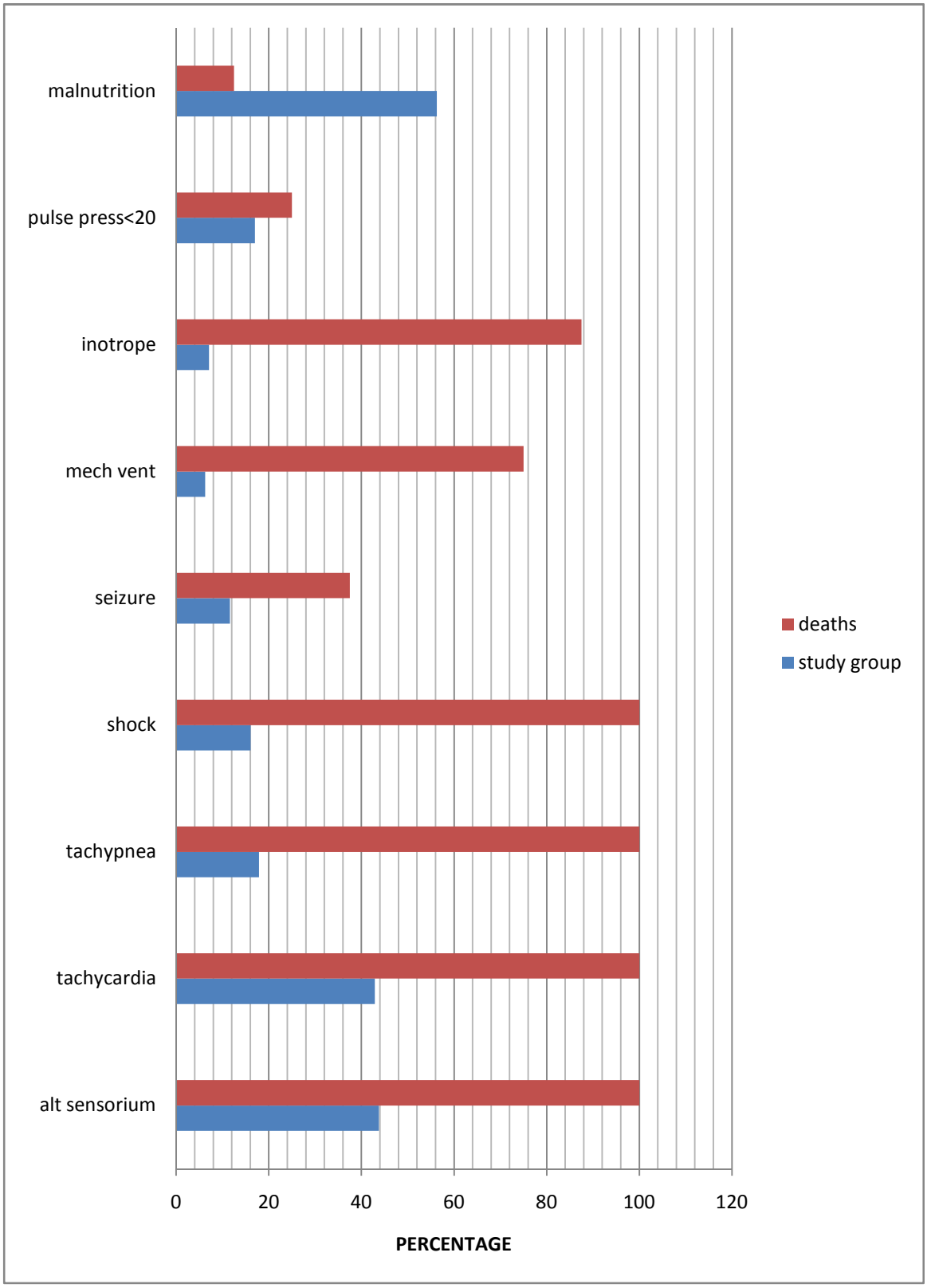
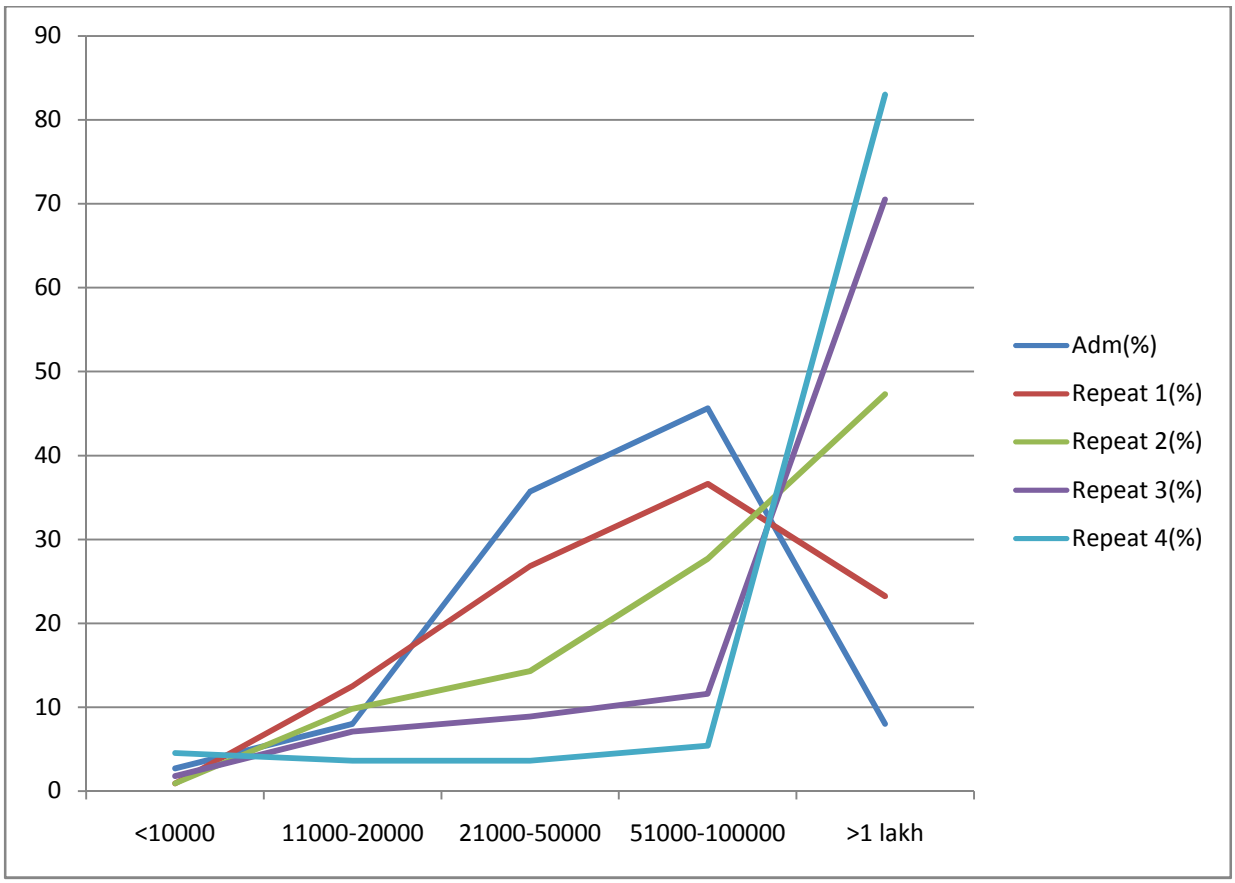


TABLE 19: PLATELET TRENDS DURING HOSPITAL STAY

Platelet	Adm (%)	Repeat 1(%) p=0.189	Repeat 2(%) p=0.004	Repeat 3(%)	Repeat 4(%)
<10000	2.7	0.9	0.9	1.8	4.5
11000-20000	8	12.5	9.8	7.1	3.6
21000-50000	35.7	26.8	14.3	8.9	3.6
51000-100000	45.6	36.6	27.7	11.6	5.4
>1 lakh	8	23.2	47.3	70.5	83

FIGURE 17:GRAPH SHOWING THE PLATELET TRENDS DURING VARIOUS STAGES OF HOSPITAL STAY



Most patients at admission(45.6%) had a platelet count in the range of 50,000-1 lakh. Single lowest count reached by a patient was 6000µ/L. Mean platelet count at admission was 61017 µ/L.Platelet trend analysis show a significant upward graph indicating that most thrombocytopenia was transient.

Among the platelet counts,the initial values were not significantly related to the mortality while the second repeat platelet value had a significant bearing on the outcome(**$p<0.05$**).Thus,rather than the absolute values,it is the drop in counts which is associated with poor outcome.

TABLE 20:TYPES OF BLEEDING MANIFESTATION

S.no.	Type of bleed	No	Percent
1	g i bleed	46	41.07
2	epistaxis	3	2.7
3	gum bleed	3	2.7
4	iv site	1	0.9
5	petechiae	11	9.8
6	purpura	6	5.4

Bleeding manifestations were seen in a total of 67 patients(59.8%).GI bleed was the commonest bleeding manifestation associated with thrombocytopenia,seen in total of 46 patients.39.3% patients had malena,20.5% of children had hematemesis.3.6% had more than one bleeding manifestation. Children with hematemesis had a significantly poor outcome(**p=0.000**) compared to children with malena(p=0.52).

TABLE 21:CORRELATION BETWEEN THE COUNTS, ERYTHEMATOUS RASH AND HESS TEST POSITIVITY

S.No.	Platelet Count	Frequency	Patients with rash	% of total	Hess test	% of total
1	<10,000	7	3	42.9	2	28.6
2	11,000-20,000	17	9	52.9	5	29.4
3	21,000-50,000	38	21	55.2	8	21.1
4	51,000-1,00,000	50	21	42	4	8
	TOTAL	112	54	48.21 (p>0.05)	19	16.96 (p>0.05)

48.2% children had a rash.Children with counts between 21000-50000 had the highest incidence of erythematous rash.Hess test was positive in 16.9% of the children in the study group.Hess test positivity was mostly seen in children with counts between 10000- 20,000(58%).But these are not significant.Both rash and Hess test are not associated significantly with the outcome(p>0.05) .Hess test was significantly positive in 23.9% children with bleeding(**p=0.017).**

FIGURE 20:COMPARISON OF BLEEDING MANIFESTATIONS

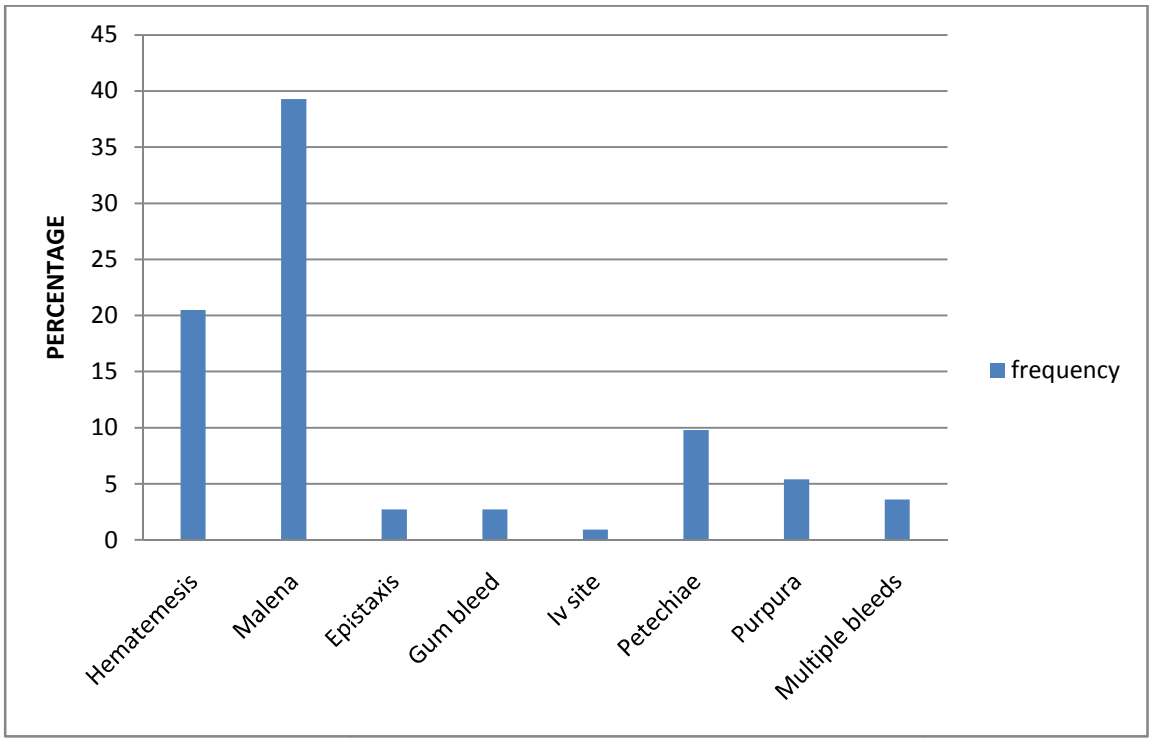


FIGURE 21:CORRELATION BETWEEN COUNTS,RASH AND HESS TEST

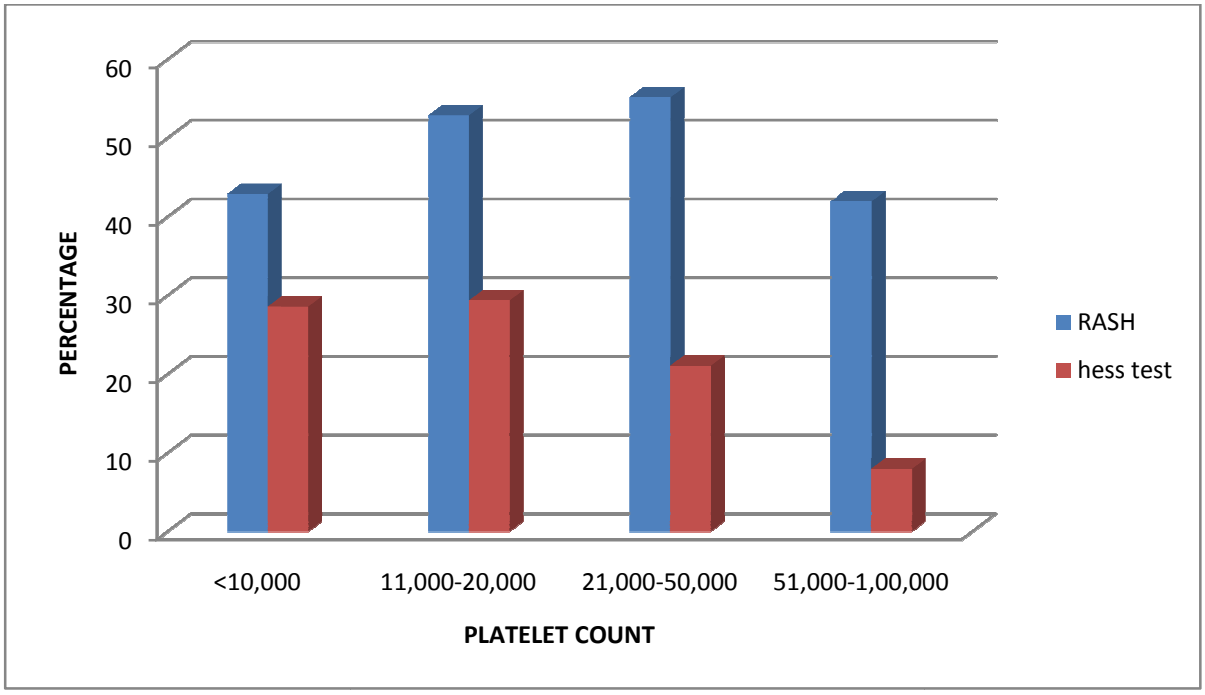
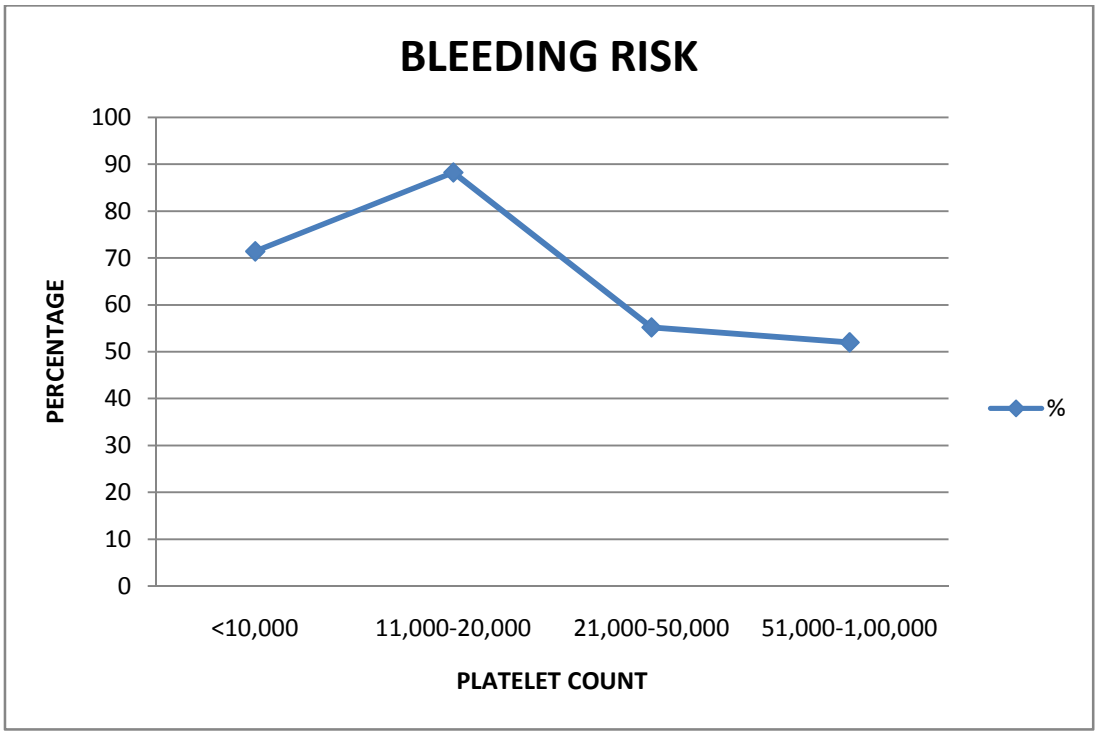


FIGURE 19:CORRELATION BETWEEN PLATELET COUNT AND BLEEDING RISK



The bleeding risk has been highest among children with platelet counts between 11,000-20,000 platelets.At counts lesser than 10,000 there has not been necessarily an increased % of bleeders.At higher counts,the percentage of bleeders is equally comparable(55.2% at counts between 21000-50000 and 52% at counts >50,000). So we can clearly see that there is poor correlation between the platelet counts and bleeding risk in the study group($p>0.05$)

TABLE 22:PATIENTS WITH BLEEDING MANIFESTATION UNDER DIFFERENT PLATELET RANGE GROUPS AND THE MORTALITY

platelet count	n as % of total	patients with bleeding	%	no of deaths	percentage mortality	mortality among bleeders in %
<10,000	6.3	5	71.4	4	57.1	80
11,000-20,000	15.2	15	88.2	3	17.7	20
21,000-50,000	33.9	21	55.2	1	2.6	4.8
51,000-1,00,000	44.6	26	52	0	0	0
TOTAL	100	67(p=0.153)	59.8	8	7.1	11.9(p=0.097)

Children with counts between 11000-20000 had the highest number of bleeding manifestations(88.2%)followed closely by children with counts less than 10,000(71.4%).Children with severe thrombocytopenia less than 10,000 had a poor outcome.They constituted 50% of the total deaths and the mortality rate was 57.1%. The mortality was higher(80%) when children with counts less than 10,000 developed bleeding manifestations.The overall mortality among bleeders is 7.1%.But none of these findings are statistically significant **.**

FIGURE 22:DISTRIBUTION OF PLATELET COUNTS IN STUDY GROUP

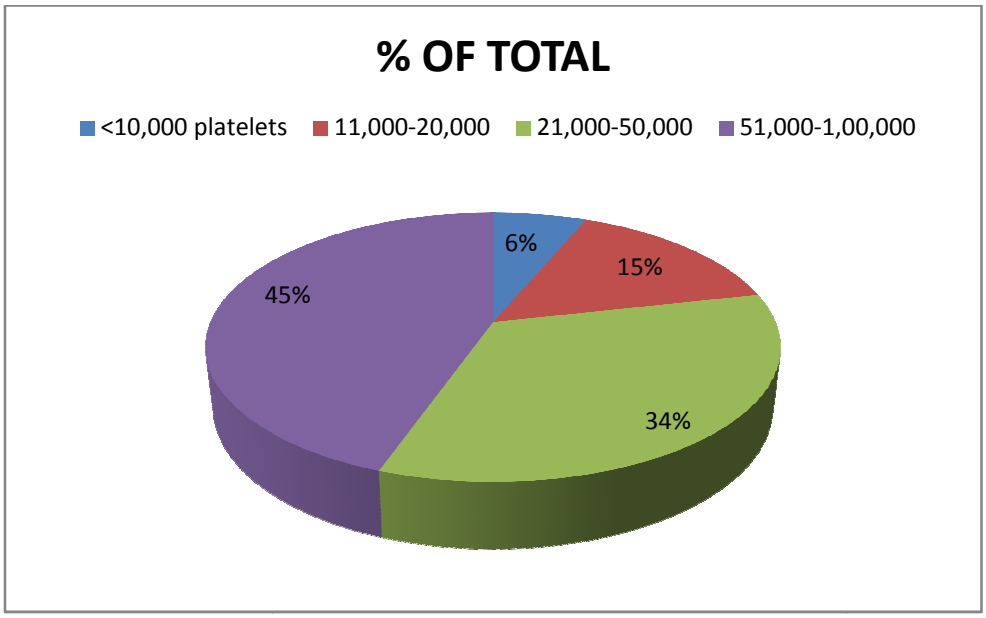


FIGURE 23:BLEEDING MANIFESTATIONS UNDER DIFFERENT PLATELET RANGES AND MORTALITY AMONG BLEEDERS

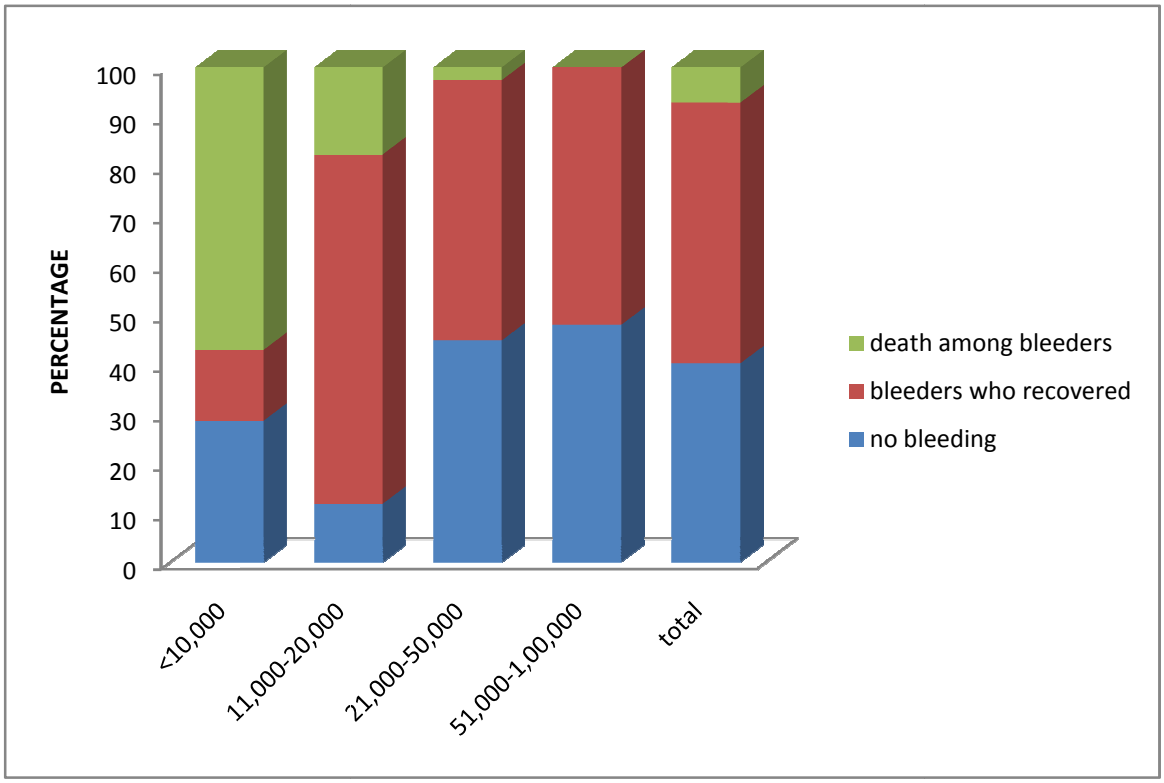


TABLE 23:COMPARISON OF THE LAB PARAMETERS

S.NO.	investigation	total n=112(% of n)	dengue n=66(% of n)	enteric n=13(% of n)	leukemia n=5	septicemia n=5(% of n)
1	Leucopenia	32(28.6)	17(25.8)	5(38.5)	2(40)	1(20)
2	Leukocytosis	29(25.9)	13(19.7)	4(30.7)	3(60)	0
3	Pancytopenia	4(3.6)	1(1.5)	0	2(40)	0
4	Anemia(**p<0.05**)	40(35.7)	15(22.7)	3(23)	5(100)	4(80)
5	Inc esr	41(36.6)	14(21.2)	7(53.9)	4(80)	4(80)
6	Inc urea/creat	27(24.1)	17(25.8)	1(7.7)	1(20)	2(40)
7	Inc liver enz	33(29.5)	15(22.7)	5(38.5)	0	0
8	Inc bilirubin	12(10.7)	2(3.03%)	1(7.7%)	0	0

Anemia and an increase in the erythrocyte sedimentation rate were the most common hematological abnormalities associated with thrombocytopenia.Enteric fever children had either leucopenia or leukocytosis and increased liver enzymes,in higher proportion compared to children with Dengue.Anemia was seen in all the leukemia cases.Increase in the ESR, azotemia had high association with septicemia.Children with anemia had a significantly poor outcome(**p=0.008**).The mean Hb in the discharged patients was 11.32gm% compared to 8.69gm% in children who expired(**p=0.007**).The other laboratory parameters did not significantly alter the outcome.

FIGURE 24: FREQUENCY OF LAB ABNORMALITIES IN THE STUDY GROUP

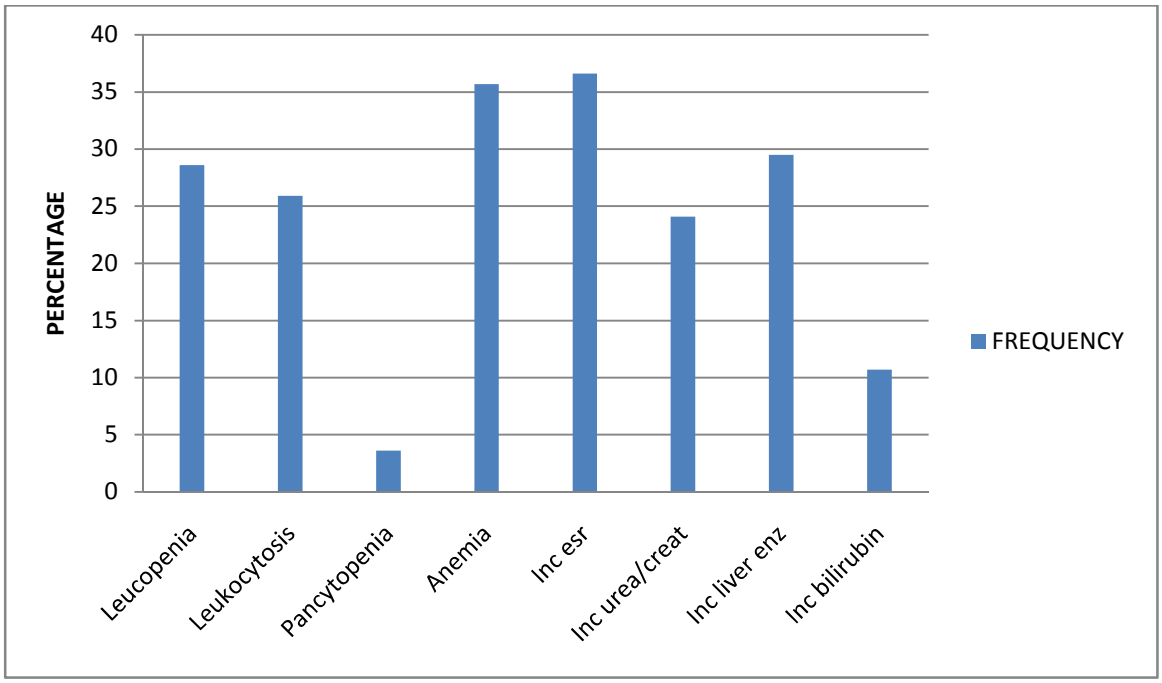


FIGURE 25:COMPARISON OF LAB ABNORMALITIES BETWEEN DENGUE AND ENTERIC FEVER

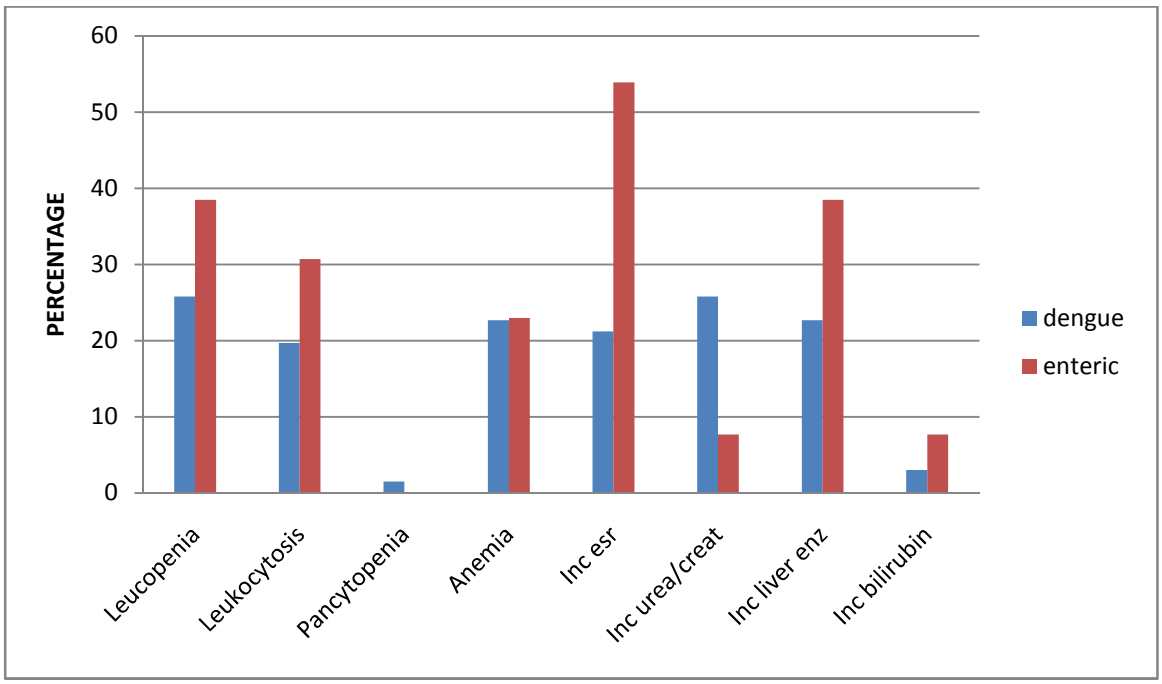


TABLE 24:COMPARISON OF PLATELET COUNTS BASED ON ETIOLOGY

Counts	dengue n=66(% of n)	enteric n=13(% of n)	D/E mixed n=4(%)	malaria n=3(% of n)	ALL n=5(% of n)	Sepsis n=5(% of n)
<10,000	2(3)	0	0	0	1(20)	1(20)
11000-20,000	10(15.2)	0	0	0	1(20)	0
21000-50,000	22(33.3)	4(30.8)	3(75)	2(66.7)	2(40)	2(40)
51000-100000	32(48.5)	9(69.2)	1(25)	1(33.3)	1(20)	2(40)

Dengue cases have had the lowest counts.Counts have not been less than 20,000 in enteric fever and malaria.Even in dengue,the commonest platelet level is more than 50,000.

TABLE 25:COMPARISON OF BLEEDING IN RELATION TO THE ETIOLOGY

Counts	patients with bleed	dengue n=66	enteric n=13	D/E n=4	malaria n=3	ALL n=5	sepsis n=5
<10,000	5	2(40)	0	0	0	0	0
11000-20,000	15	9(60)	0	0	0	1(6.67)	0
21000-50,000	21	11(52.4)	2(9.5)	2(9.5)	1(4.8)	1(4.8)	1(4.8)
51000-100000	26	14(53.9)	6(23.1)	1(3.9)	0	1(3.9)	2(7.8)
Total	67	36(54.6)	8(61.5)	3(75)	1(33.3)	3(60)	3(60)

Among bleeders too,the most common etiology was Dengue fever.But patients with enteric fever and thrombocytopenia had higher incidence of bleeding compared to even dengue cases.In fact,co infection with both diseases had the highest incidence of bleed(75%).In children with Dengue,counts less than 20,000 had high association with bleeding,while in enteric fever there was no such correlation with the counts for the predisposition to bleed.

FIGURE 26:COMPARISON OF THE PLATELET COUNTS BETWEEN DENGUE AND ENTERIC FEVER

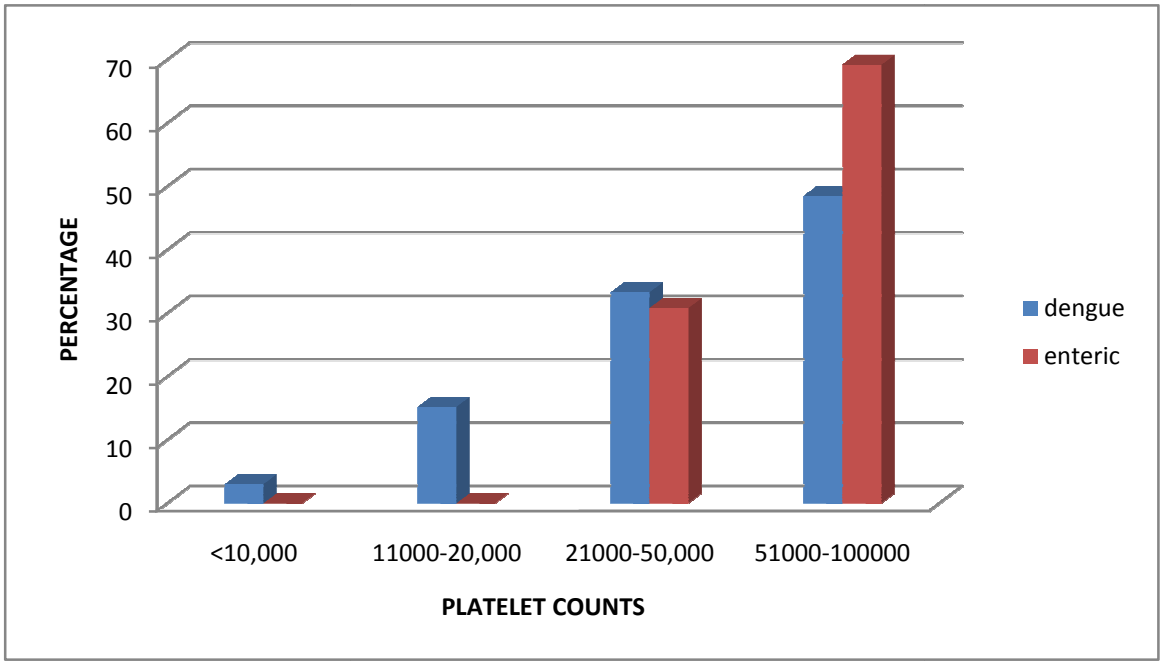


FIGURE 27:COMPARISON OF BLEEDING MANIFESTATIONS BETWEEN DENGUE AND ENTERIC FEVER

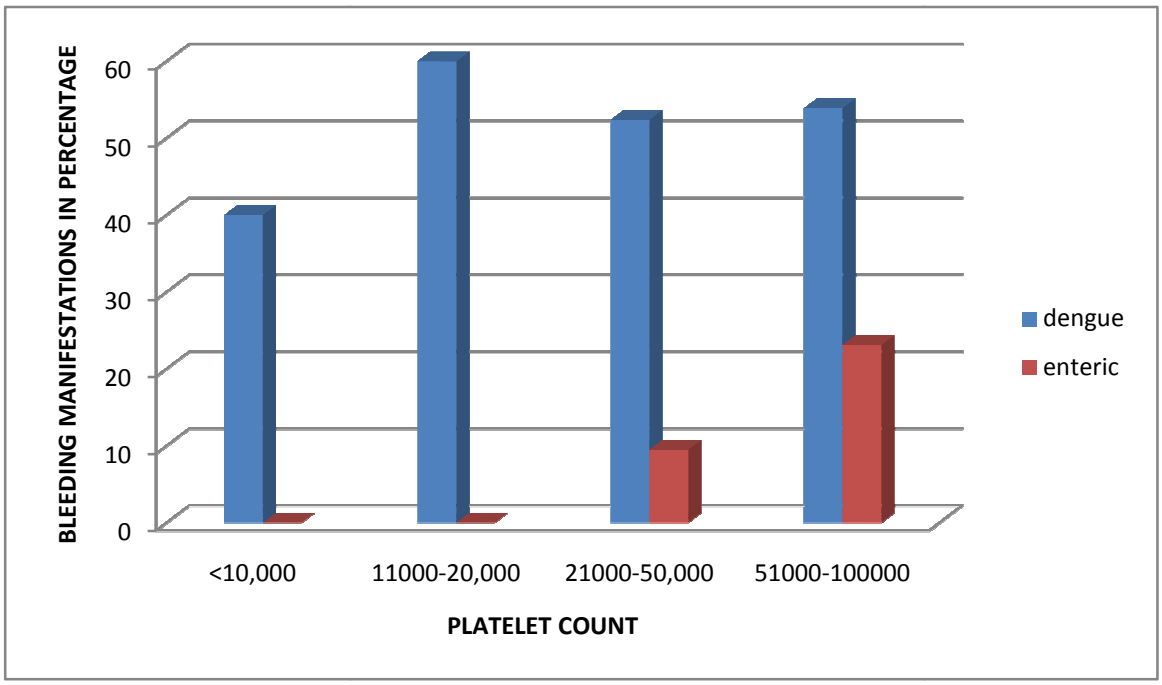


FIGURE 28:DISTRIBUTION OF PLATELET COUNTS IN DENGUE FEVER

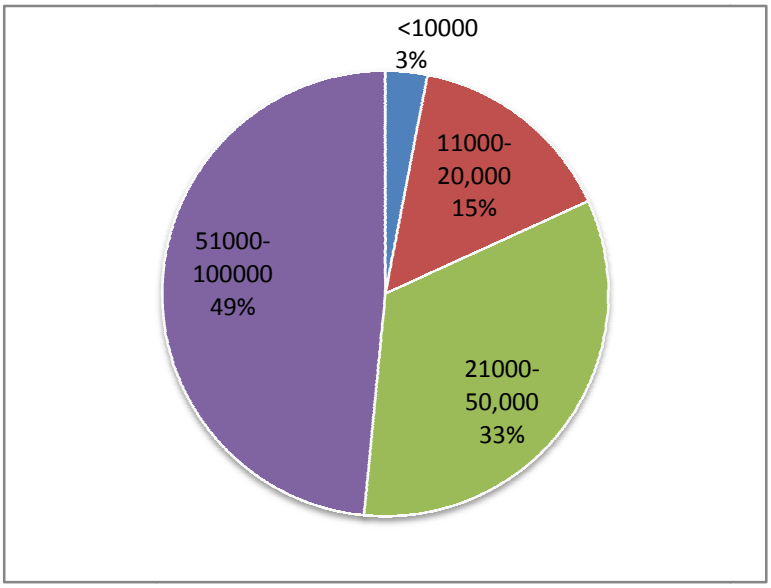


FIGURE 29:DISTRIBUTION OF BLEEDING MANIFESTATIONS IN DENGUE FEVER

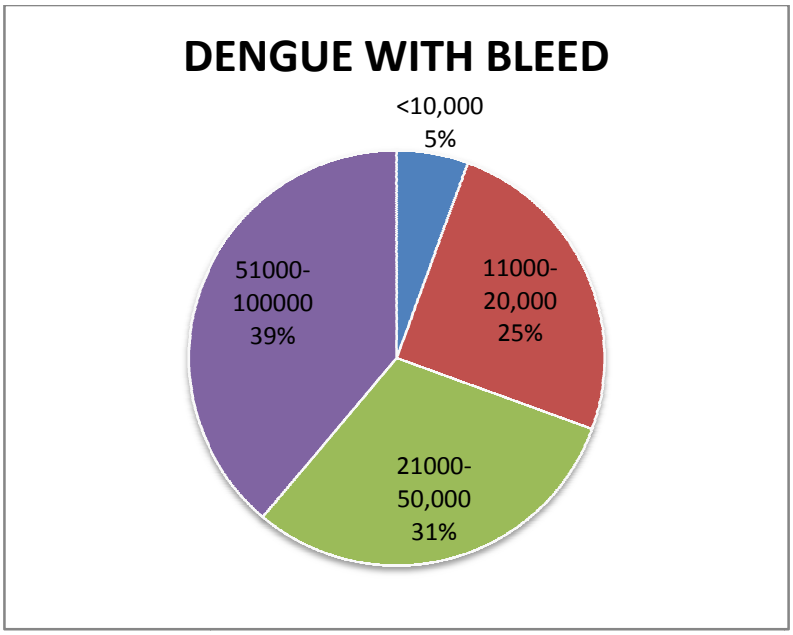


TABLE 26:COMPARISON OF THE THREE TYPES OF DENGUE WITH RELATION TO THE PLATELET COUNTS

S.NO.	COUNTS	DF (n=36)	DHF (n=21)	DSS (n=9)
1	<10,000	0	0	2(100)
2	11000-20,000	2(20)	4(40)	4(40)
3	21000-50,000	11(50)	8(36.4)	3(13.6)
4	51000-100000	23(71.9)	9(28.1)	0

Among dengue cases,counts less than 10,000 had a 100% incidence of Dengue shock syndrome.Most patients with Dengue shock syndrome had counts between 11000-20,000(40%).Platelet counts less than 20,000 have thus had a high association with severe dengue.

TABLE 27:COMPARISON OF THE THREE TYPES OF DENGUE WITH RELATION TO BLEEDING

S.NO.	counts	dengue with bleed	DF(n=36)	DHF(n=21)	DSS(n=9)
1	<10,000	2	0	0	2(100)
2	11000-20,000	9	1(11.1)	4(44.4)	4(44.4)
3	21000-50,000	11	5(45.5)	5(45.5)	1(9.1)
4	51000-100000	14	8(57.1)	6(42.9)	0
	Total	36	14(11.1)	15(71.4)	7(77.8)

DSS children had a higher incidence of bleeding manifestations (77.8%),and the bleeding risk in DSS was high when the counts were below 10,000.Comparatively only 11.1% of dengue fever cases had bleeding.In DHF too,the incidence of bleeding manifestations was high(71.4%) but there was no correlation with lower counts to occurrence of bleeding manifestations.

FIGURE 30: COMPARISON OF PLATELET COUNTS IN DENGUE

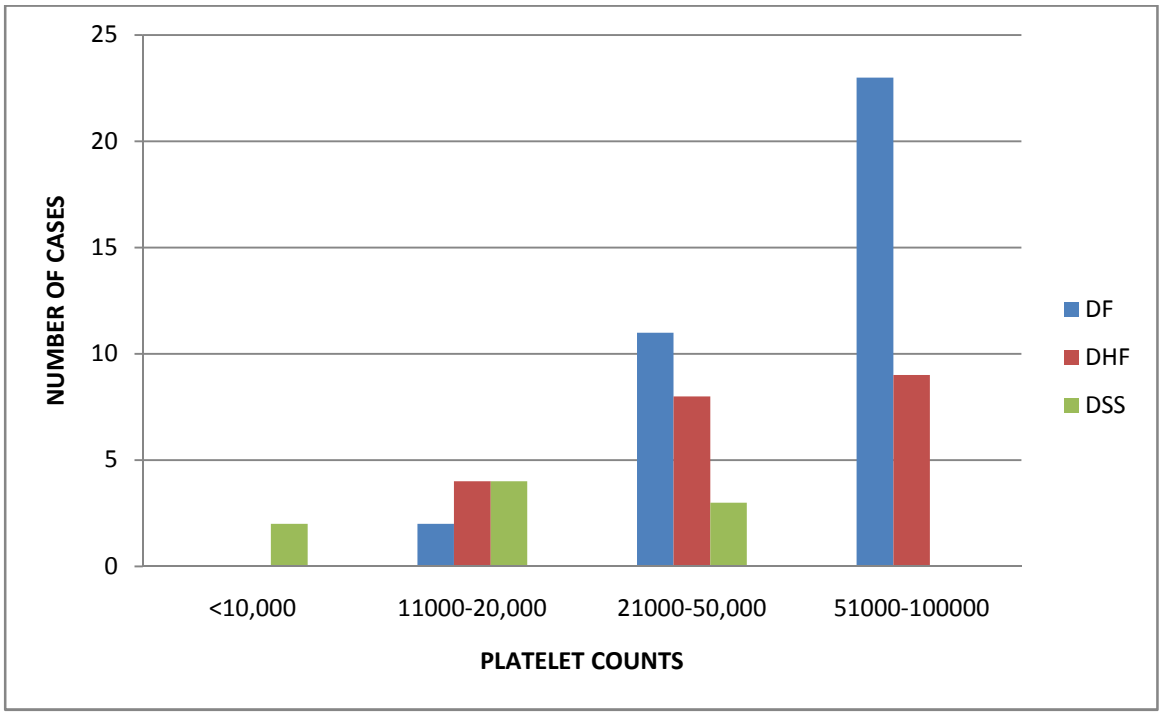


FIGURE 31:COMPARISON OF THE INCIDENCE OF BLEEDING MANIFESTATIONS IN DENGUE

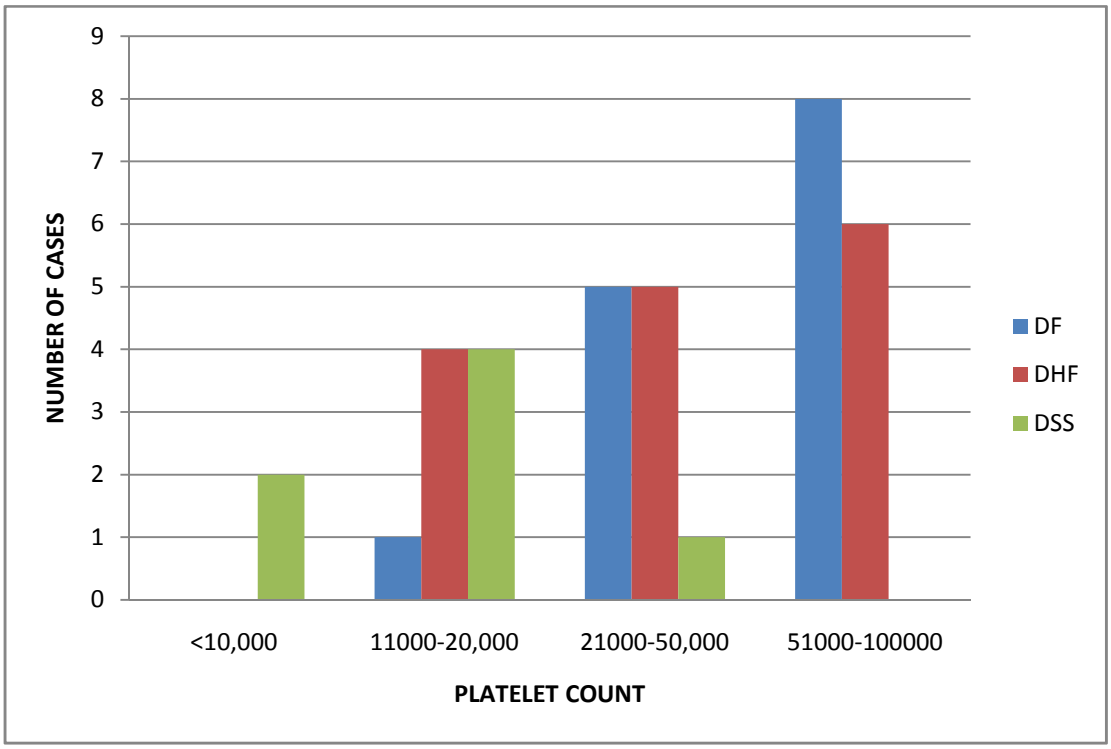


TABLE 28:COMPARISON OF MEAN HEMATOLOGICAL VALUES BASED ON ETIOLOGY

s.no.	Diagnosis	Mean Hb(gm%)	Mean PCV(%)	Mean platelet count
1	DF	11.93	37.74	60666
2	DHF	11.98	39.77	46714
3	DSS	11.21	38.04	18000
4	enteric	12.15	35.58	62076
5	ent/den co inf	11.38	35.55	41500
6	malaria	11.03	34.47	46333
7	leukemia	4.27	13.36	35600
8	septicemia	9.96	30.56	50800
	Total(n=112)	11.13	35.99**(p=0.016)**	48250

The mean hemoglobin value in the study group is 11.13gm%.The mean hematocrit value is 35.99%,and the outcome is significantly poor among children with hematocrit below the mean**(p<0.05)** probably indicative of the mortality risk associated with bleeding .The mean platelet count is 48,250/µL.Hematocrit was highest among DHF(39.77) cases with slight decrease in DSS cases(38.04).The mean platelet count was significantly lower in DSS cases(18,000/µL).Anemia with thrombocytopenia was common in leukemia and septicemia(4.27 gm% and 9.96gm% respectively).

TABLE 29:COMPARISON OF THE DENGUE SEROPOSITIVITY

S.NO.	Dengue panel	DF (n=36)	DHF (n=21)	DSS (n=9)	Enteric/deng co inf(n=4)
1	IgM POSITIVE	8(22.2)	4(19)	7(77.8)	0
2	IgG rise in titre	5(13.9)	0	0	1(25)
3	NS1 POSITIVE	5(13.9)	0	0	0
4	IgM/IgG POSITIVE	8(22.2)	10(47.60)	1(11.1)	3(75)
5	IgM/NS1 POSITIVE	7(19.4)	3(14.3)	0	0
6	TRIPLE POSITIVE	3(8.3)	4(19)	1(11.1)	0

$p>0.05$

Dengue fever cases had more monopositivity(50% cases) and IgM/NS1 positivity compared to DHF and DSS cases,indicating that they were most probably primary infection.DHF cases had a higher proportion of IgM/IgG and triple dengue panel(NS1,IgM,IgG) test positivity,suggesting that they were more of secondary infections.In contrast,DSS cases had high frequency of IgM monopositivity.

However,seropositivity in dengue is not significantly related to the outcome.(all the three investigations had p values more than 0.05).

FIGURE 32: COMPARISON OF MEAN HEMATOLOGICAL VALUES

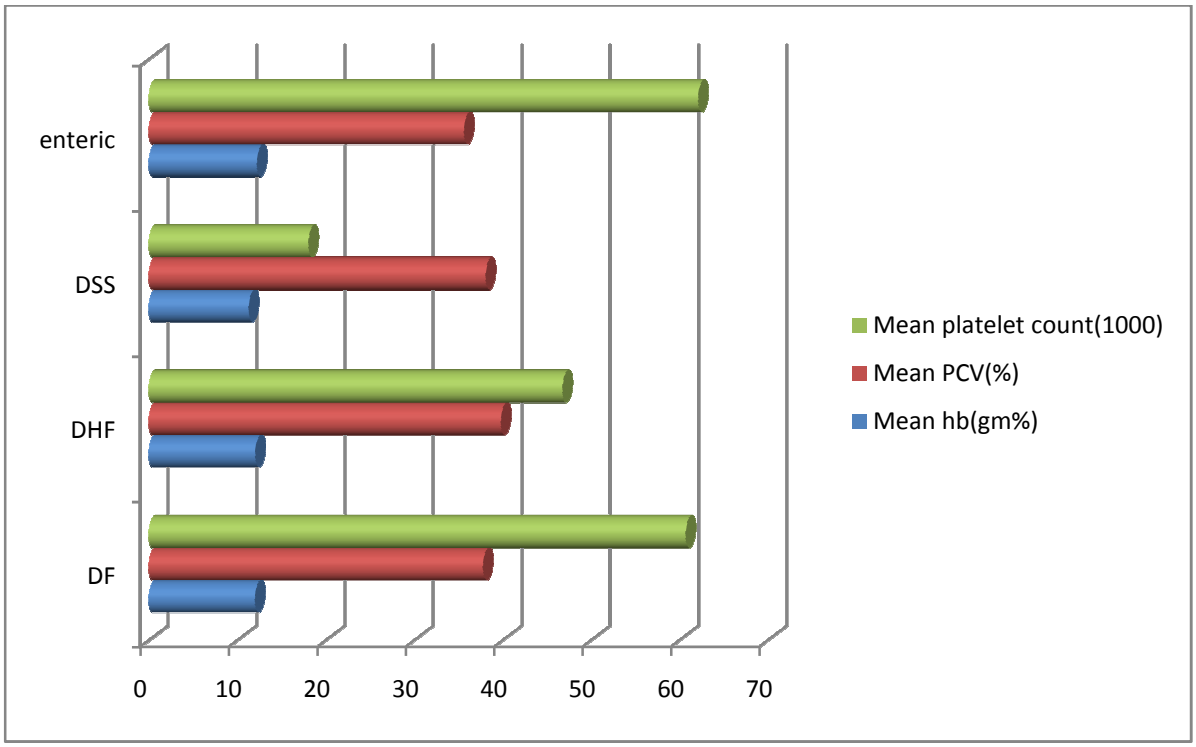


FIGURE 33:GRAPH COMPARING SEROPOSITIVITY IN DENGUE

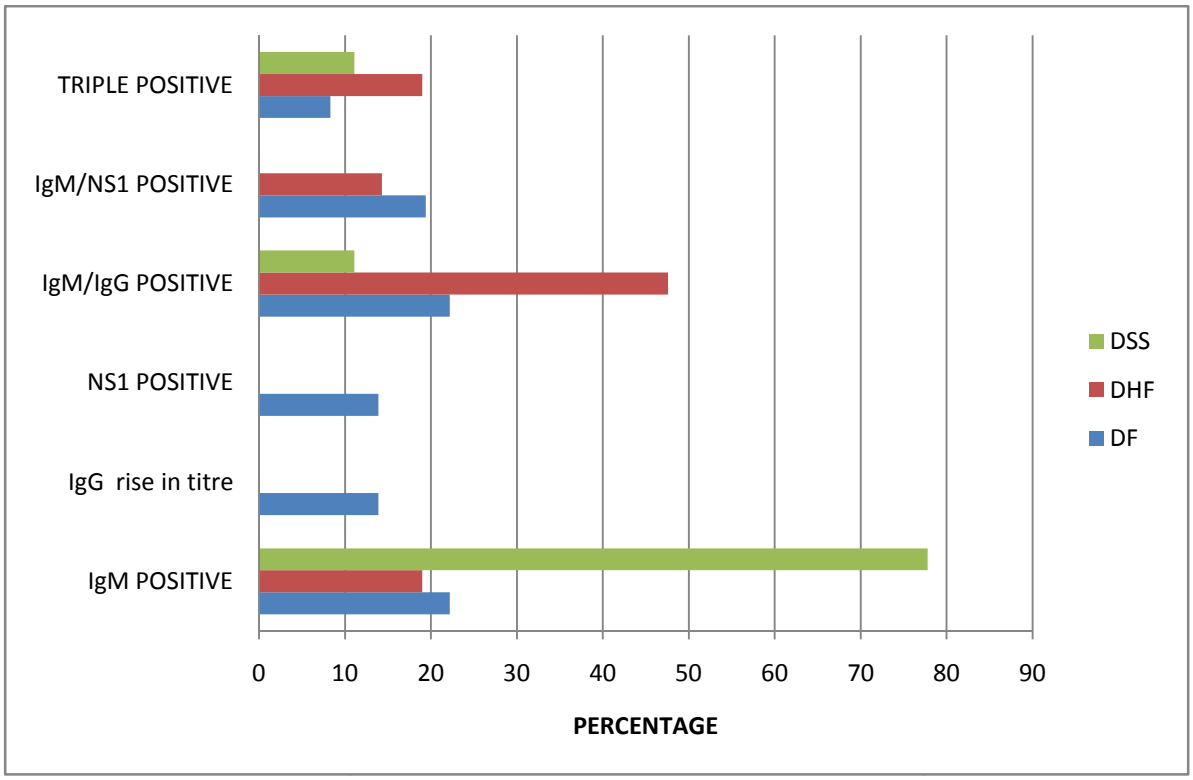


TABLE 30: COMPARISON OF RADIOLOGICAL ABNORMALITIES

Features	TOTAL (n=112) (%)	p value	DF (n=36)	DHF (n=21)	DSS (n=9)	Enteric (n=13)
X ray eff/pneumonia	15(13.4)	**0.002**	1(6.6)	4(26.6)	1(6.6)	2(13.3)
Gall bladder edema	31(27.7)	**0.001**	0	11(52.4)	4(44.4)	8(61.5)
Pleural effusion	29(25.9)	**0.008**	0	15(71.4)	5(55.5)	4(30.7)
Ascites	18(16.1)	0.062	0	8(38.1)	3(33.3)	3(23.1)
Hepatomegaly	45(40.2)	0.338	10(27.8)	9(42.9)	3(33.3)	10(76.9)
Splenomegaly	27(24.1)	0.397	6(16.7)	5(23.8)	0	4(30.7)

Abnormal X ray findings had a significant association with mortality(**p<0.05**). Ultrasound abdomen was a very useful radiological tool among the study population.It was able to pick up features of polyserositis with high sensitivity in both DHF and DSS.But the findings were not specific for dengue alone.Even in enteric fever,pericholecystic edema with gall bladder thickening was a consistent feature(61.5%).Hepatomegaly was seen in more than 75% of both enteric fever and enteric/dengue co infection cases.Gall bladder wall edema and pleural effusion in Ultrasound were seen in high frequency in children with bleeding.This is statistically significant(**p<0.05**).

FIGURE 34:FREQUENCY OF ULTRASOUND FINDINGS IN CHILDREN WITH THROMBOCYTOPENIA

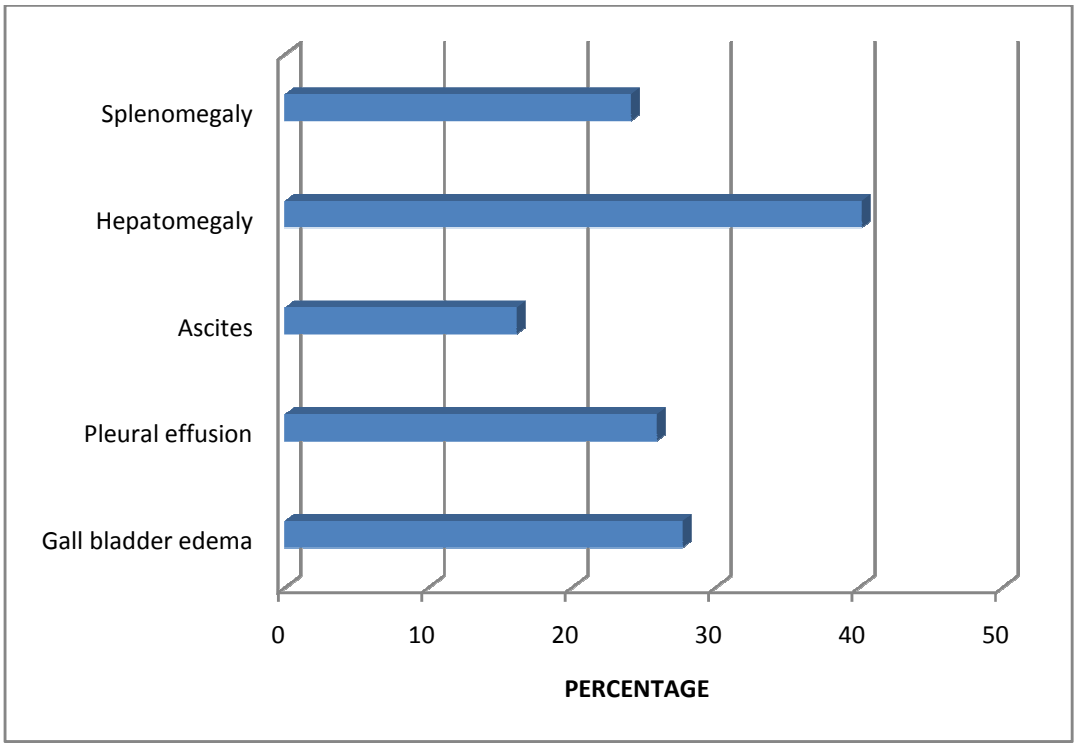
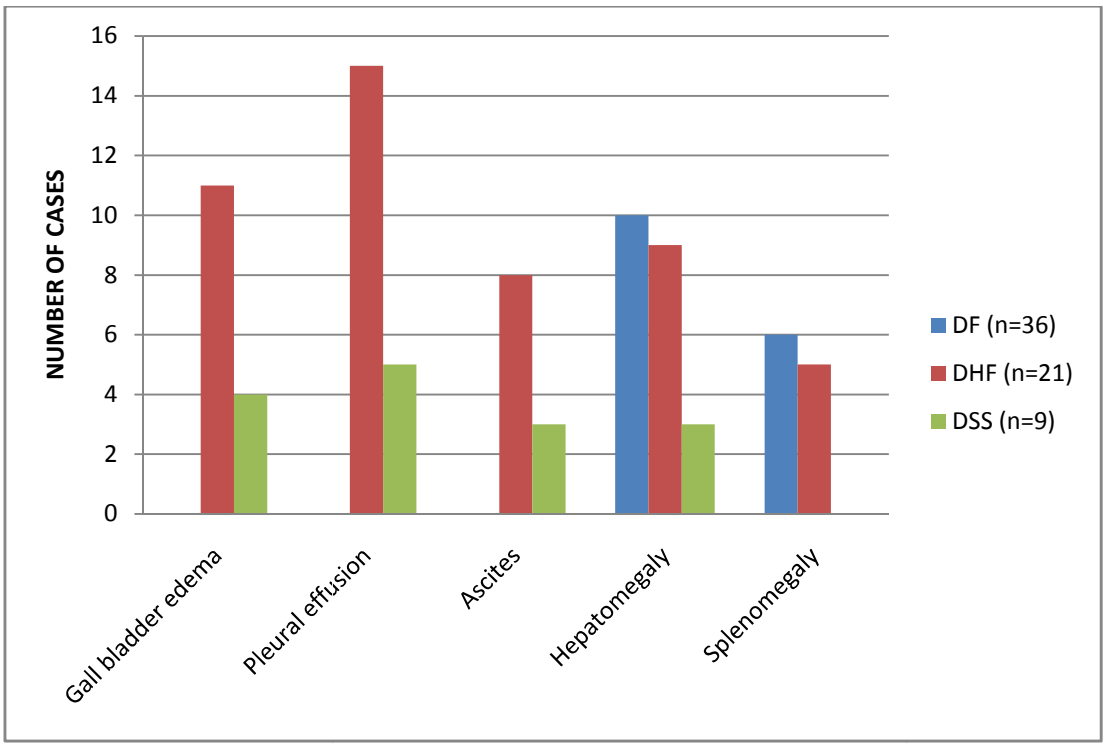


FIGURE 35: COMPARISON OF THE ULTRASOUND FINDINGS IN DENGUE



TRANSFUSION REQUIREMENTS

Standard protocols were followed in transfusing blood products to the children.Majority received packed cells and platelets,while lesser number of patients were transfused whole blood and colloids.The cause for most transfusions was severe shock with bleeding manifestations,while few children were transfused prophylactically in view of drastically decreasing counts.

TABLE 31:PROFILE OF TRANSFUSED CHILDREN

Parameter	**no of children**	**%**
Children who received blood products	20	17.9%
Children who had bleeding	17	85%(**p<0.05**)
Mortality among transfused patients	6	30% (**p<0.05**)
Children with associated anemia	15	75%(**p=0.001**)
Lowest count for which transfused	7000	
Mean transfusion volume requirement	43.75 ml/kg/patient	
Mean count of transfused patients	24,000/L(**p=0.038**)	
Major diagnosis for which transfused	DSS	
% of DSS patients transfused	77.8%	

Anemic patients with thrombocytopenia had significantly increased need for transfusions(**p<0.05**).The mean Hb of transfused patients was 9.65gm% compared to 11.46gm% in non transfused patients(**p=0.006**).

3 patients who were transfused had counts less than 10,000/µL.9 children had counts between11,000-20,000/µl,7 between 21,000-50,000/µl while one patient with a count of 78000 was transfused.72.9% patients who had low platelet counts were not given any transfusion and improved.This is statistically significant(**p=0.007**).Also patients with low admission platelet counts and also rapid drop in counts required more transfusions (**p<0.05**).

25.4% of patients with bleeding manifestations were transfused(**p<0.05**). Among the bleeding manifestations,children with hematemesis and malena had a significantly increased need for transfusions(**p<0.05**).

There was also statistically significant association between need for transfusion and mortality(**p<0.05**).All the poor predictors of mortality also had significant association with the need for transfusions.Hence,it can be implied that transfusions have not significantly altered the outcome.

DISCUSSION

6. DISCUSSION

As discussed in the literature,thrombocytopenia,being associated with bleeding manifestations is now considered an independent parameter predicting outcome in the paediatric intensive care unit[64]. Here,critical analysis of the observations of our study is performed, comparing it with other Indian and foreign studies.

AGE WISE INCIDENCE:

Analysis of our study shows the highest incidence of thrombocytopenia in the 6-10 years age group,with a mean age of 6.56 years.Ali jan et al[59] found higher incidence in the 1-5 yrs age group,while Sachdev et al[60] reported two and a half years as the median age.The present study is comparable with Shahanaz et al,Lahore which reported 4-7 years as the commonest age group affected.

SEX WISE DISTRIBUTION:

There is no particular sex predilection for thrombocytopenia.The male female ratio in the present study was 0.89:1 while Sachdev et al[60] reported 1.76:1 and Ali jan et al[59] reported 1.9:1.Such wide variations could be due to socio cultural differences.

INCIDENCE:

The incidence of thrombocytopenia has been quoted to vary from 13-58% in various studies[2].The present study has shown 15.95% incidence,which is comparable to other studies;Sachdev et al[60] showing

23.2% and 22% in a neonatal ICU[65].A study done in St.louis university,USA by Krishnan et al also showed a very similar incidence of 17.3%.

PLATELET TRENDS:

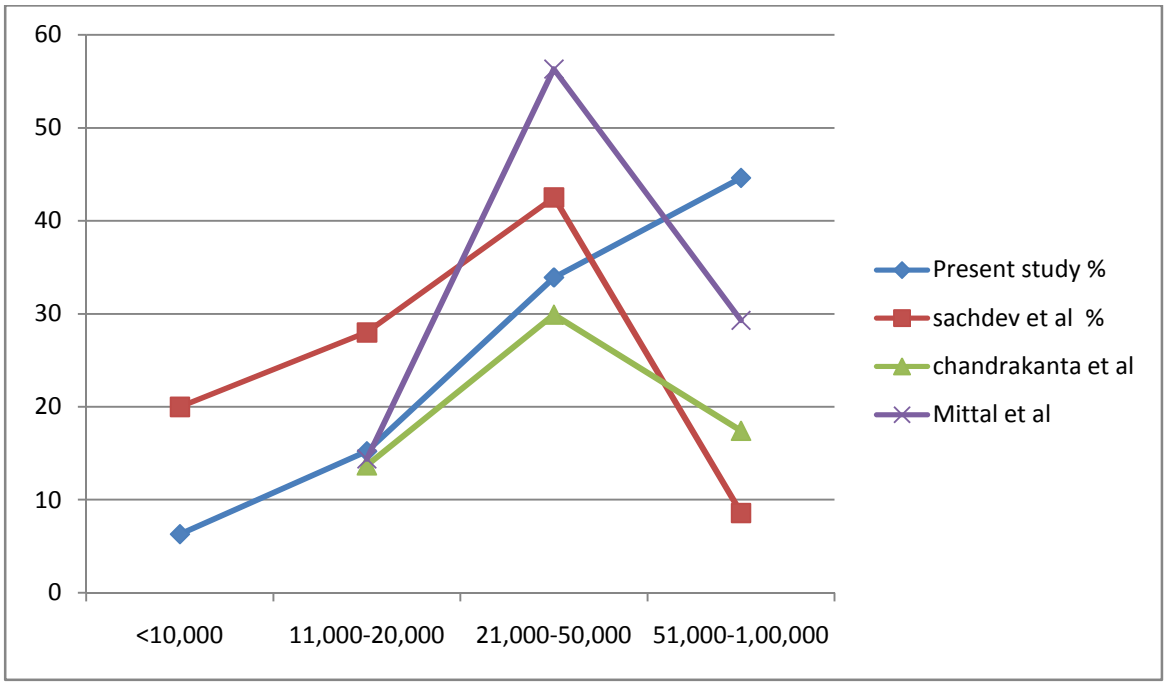
The present study had significantly less number of children with counts less than 10,000 compared to Sachdev et al[60](20%).But it is comparable in the other platelet ranges(15.2% between 11,000-20,000) to studies by Chandrakanta et al[66](13.7%) and Mittal et al[62],University College of Medical Sciences,Delhi(14.4%).

TABLE 32:COMPARISON OF PLATELET TRENDS BETWEEN VARIOUS STUDIES

S.NO.	platelet count	Present study %	sachdev et al[60] %	Chandrakanta et al[66]	Mittal et al[61]
1	<10,000	6.3	20		
2	11,000-20,000	15.2	28	13.7	14.4
3	21,000-50,000	33.9	42.5	29.9	56.3
4	51,000-1,00,000	44.6	8.6	17.4	29.3

Children with counts less than 10,000 had the worst outcome(57.1% mortality).Drop in platelet count >27% was significantly associated with mortality reported by Sachdev et al,which is comparable to the findings of the present study.

FIGURE 36: COMPARISON OF PLATELET COUNT TRENDS BETWEEN THE VARIOUS STUDIES



MORTALITY:

Mortality in the present study was 7.1% with DSS contributing to 50% of the deaths.Krishnan et al[64] reported 17.1% mortality,while Sachdev et al[60] reported a mortality of 10.9%. Mortality was highest among infants(45.5%) and younger chidren(<3 years).This is consistent with the observation by WHO[67] that infants are prone for severe forms of the disease. The WHO observed case fatality rate in India is 3-5%.

ETIOLOGY COMPARISON:

The commonest etiology for newly diagnosed thrombocytopenia among children in the present study was Dengue(58.8%).Ali jan et al[59] reported ITP as the commonest diagnosis(32%).

TABLE 33:COMPARISON OF ETIOLOGY BETWEEN STUDIES

S.no.	Etiology	Nair PS et al, St. Stephen's Hospital, , 2003[68](n=109)	Present study(n=112)
1	Dengue	13.8%	58.8%
2	Enteric fever	14.7%	11.6%
3	Septicemia	26.65%	4.5%
4	Malaria	9.2%	2.7%
5	malignancy	3.7%	4.5%
6	Others/unknown	18.3%	14.2%

The variation in the etiological data could be due to the differences in disease prevalence between the northern and southern parts of the country.Also,the study was done during the dengue season,explaining the higher percentage of dengue cases.

COMPARISON OF DENGUE DATA:

Since dengue was the commonest diagnosis in the present study population with a good sample size(66cases),a detailed analysis of dengue was performed.Dengue fever with or without hemorrhage(DF) was most common(32.1%) . DSS comprised only 8% of cases with

thrombocytopenia, but had the highest mortality rate of 44.4%.This could be attributed to the high baseline microvascular permeability in children[69].

Abdominal pain and vomiting were present in half to two-third of the patients in the present study which is consistent with other studies.Similar to other studies,platelet counts had no correlation to the bleeding manifestations in dengue.Majority of bleeding in the present study was observed in patients with counts more than 50,000.This confirms further that bleed in dengue is due to aberrant immunity and cross reactivity rather than a effect of the decrease in number.Indiscriminate platelet transfusions thus need to be avoided.

DSS cases with counts less than 10,000 have had a higher incidence of bleeding manifestations and mortality.Intensive monitoring and judicious fluid management may be needed to improve the outcome in such children.

Gomber et al[70] defines 36.3% as the cut-off hematocrit for DHF.In the present study,the mean hematocrit among dengue patients was well above this cutoff,indicating high predisposition for development of severe dengue in the study group.Also,the hematocrit values are significantly associated with mortality.Unlike previous studies,the incidence of co infections in the present study was low(5.7%).But the children with dengue/enteric co infection had abdominal tenderness in a higher proportion.The mortality rate among dengue patients as a whole was low(4/66=6.06%) .

Erythematous rash with flushing was very commonly observed in children with dengue in the present study(70.4%).This is inspite of the dark complexion in our children.Hence,there must be a high index of suspicion regarding dengue whenever a child presents with fever and rash in dengue endemic area.The present study showed a higher incidence of bleeding manifestations compared to previous studies but less number of severe forms of the disease(DHF and DSS).This could probably be due to the serotype of the dengue virus that is circulating in and around Tirunelveli, and also the environmental factors.Serotyping could not be done in the present study,which could be a prospective area for further studies.

TABLE34:COMPARISON OF STUDIES ON DENGUE

Author	**Chandrakanta et al[66]**	**Mittal et al[61]**	**Present study**
Place	Lucknow	Delhi	Tirunelveli
DF/DHF/DSS	31.2% DHF	8/51/42	54.6/31.8/13.6
Fever	100%	100%	100%
Abdominal pain	25%	71%	53%
Vomiting	41.2%	-	63.6%
Bleeding	38.8%	48.8%	54.6%
Rash	37.5%	26.6%	70.4%
Hypotension	10%	43%	13.6%
Hepatomegaly	62.5%	31.1%	33.3%

Author	Chandrakanta et al[66]	Mittal et al[61]	Present study
Splenomegaly	60%	27%	16.7%
Leucopenia	-	17.7%	25.8%
Mean hct	26.8%	36.1%	38.5%
Mean hb	9.8gm	11.5gm	11.7gm
Plt <20,000	13.7%	14.4%	18.2%
20-50,000	29.9%	56.3%	33.3%
>50,000	17.4%	29.3%	48.5%
Deranged LFT	66.7%	48%	22.7%
Pleural effusion	-	6%	30.3%
Ascites/gb edema	-	13%	22.7%

HESS TEST:

Hess test was positive in 16.9% of the children in the study group.Hess test positivity was mostly seen in children with counts between 10000- 20,000(58%).85.7% positivity was shown by Shigeki Hanafusa (Japan) et al[71],23.7% by Narayanan et al and 52% by Kalyanarooj et al (Indonesia).The low percentage in the present study(similar to another Indian study Narayanan et al)compared to foreign studies may be due to the difference in skin complexion and capillary fragility in Indian children.Hess test in the present study had significant association with bleeding.

SEROLOGICAL INVESTIGATIONS IN DENGUE:

The present study showed 63.9% NS1 positivity,84.8% IgM positivity and 48.5% showed fourfold rise of IgG.Comparatively a study in Kuala lumpur Malaysia by Kassim et al[72] showed 32.2% NS1 positivity,40.9% IgM and 36.1% IgG positivity.All three investigations were very useful in identifying both primary and secondary dengue infection.Triple positivity was seen in 36% of the cases.DHF had features suggestive of secondary infection.

PREDICTORS OF MORTALITY:

Altered sensorium,tachycardia,tachypnea,shock(all having 100% association with death),requirement of inotrope support(87.5%),mechanical ventilation(70%) ,seizures,malnutrition were all significantly associated with increased mortality.Requirement of mechanical ventilation in Sachdev et al[59] was 23.9% whereas in the present study,it was 6.3%.The incidence of shock was 17.3% in Sachdev et al while in the present study it was 16.1%.

In the study group,cases with seizures and thrombocytopenia had high mortality.This could probably be because of intracranial bleed induced by the thrombocytopenia.Abdominal distension and pedal edema were significantly seen in increased frequency in children who expired.This could be probably because of plasma leakage associated with thrombocytopenia in dengue,and probable unidentified compartment

syndrome.Hematemesis was also seen in significantly increased frequency in the expired children.Children with anemia also had a significantly poor outcome probably because they could not tolerate the bleeding.

BLEEDING MANIFESTATIONS:

Bleeding manifestations were seen in a total of 67 patients(59.8%) in the present study compared to 19.5% in Sachdev et al.GI bleed was the commonest bleeding manifestation associated with thrombocytopenia,seen in total of 46 patients. There was significant association between vomiting ,abdominal distension and bleeding .Children with counts between 11000-20000 had the highest number of bleeding manifestations(88.2%)followed closely by children with counts less than 10,000(71.4%).The overall mortality among bleeders is 7.1%.Bleeders with counts less than 10,000 had 87.5% mortality.But bleeding in thrombocytopenia was not significantly related to the platelet count or to the mortality.

TABLE 35:COMPARISON OF BLEEDING MANIFESTATIONS BETWEEN VARIOUS STUDIES

S.NO.	Study	Present study	Nair et al[68]	Ali jan et al[59]
1	GI bleed	41.07%	9.2%	6%
2	Epistaxis	2.7%	6.4%	34%
3	Gum bleed	2.7%	5.5%	28%
4	Petechiae/purpura	9.8%	9.2%	32%
5	Others	0.9%	3.7%	10%

The wide variations observed are due to the different study populations.Ali jan et al[59] had more ITP cases.In ITP,milder mucosal bleeds are more common.Again,petechiae and purpura are more likely to be missed in the Indian population because of the dark complexion in our children.

TRANSFUSION REQUIREMENTS:

Inspite of several studies,evidence based guidelines for transfusions in children with thrombocytopenia are ambiguous.As discussed in the literature,in most clinical situations,the treating physician is made to act on his own discretion rather than strictly follow guidelines.

17.9% children in the present study required transfusions.They received 43.75 ml/kg/patient.In comparison,21.9% patients were transfused in the study by Sachdev et al. and transfusions were reported to be

significantly associated with mortality.Most of the patients transfused in our study were DSS patients(77.8%).The mean platelet count of transfused patients was 24,000.Yet 80% mortality was seen in the transfused patients,which was also statistically significant.Patients with low platelet counts and bleeding manifestations also did not show statistically significant improvements in comparison to non transfused patients.

Hence, the role of prophylactic platelet transfusions is to be questioned until uniform guidelines are established.WHO advises that platelet transfusions are to be avoided in dengue.The only clinical situation where platelet transfusion is needed in dengue is when the counts are less than 10,000.

The limitations in our study have already been listed.Considering that our study was a small scale study done in an area with low malaria endemicity(a disease very commonly associated with thrombocytopenia)and with serological studies for viral markers not being done ,the observations of the present study are definitely subject to confounding.There were also very few studies on paediatric thrombocytopenia for comparison.

CONCLUSION

7. CONCLUSION

- Thrombocytopenia is common in the paediatric age group.Approximately,one in every six hospital admissions develop thrombocytopenia.
- Children less than 3 years with thrombocytopenia have a poor prognosis.Therefore,they need intensive monitoring.
- Dengue fever is the leading cause for newly detected thrombocytopenia in this study.
- Dengue shock syndrome is the leading cause of mortality in the present study.Platelet counts less than 10,000 and bleeding in children with shock have the worst outcome.Judicious fluid management strictly following the WHO guidelines along with intensive clinical monitoring can go a long way in reducing mortality in this group of patients.
- Early identification of thrombocytopenia at the primary and secondary levels with proper referral do affect the outcome.Hence the need to sensitise those clinicians at these levels and strengthen these services.
- Erythematous rash with flushing is a sensitive clinical sign to identify at risk children.It needs to be looked for in children in endemic areas during seasonal outbreaks.
- Altered sensorium, tachycardia, tachypnea, shock, seizures, malnutrition, need for inotrope support and for mechanical ventilation are all predictors of mortality.Intensive care facilities at tertiary level

needs to be strengthened further in order to reduce mortality in these patients.

- Hematemesis and abdominal distension are significantly associated with mortality.They are warning signs and are to be given due importance as third space blood loss can go unnoticed and produce compartment syndrome.
- Intracranial bleed needs to be managed aggressively and appropriately.Occurence of seizures in septicemia children with thrombocytopenia has had a 100% mortality.
- Platelet counts less than 20,000 have been frequently associated with bleeding.Counts less than 10,000 had higher mortality and association with DHF and DSS.Serial platelet counts are good predictors of disease progression in these patients,because of the dynamic nature of the platelet count.
- Hess test and hematocrit done early are sensitive in picking up cases prone for severe dengue and must be done in all cases.Hess test especially is sensitive in picking up children prone for bleeding manifestations.
- Likewise,Chest X ray and Ultrasound abdomen are very sensitive in picking up polyserositis in DHF which must be done in all cases.Clinicians can be easily trained in performing an ultrasound and it is a comparatively cost effective diagnostic tool,which may be made

mandatory in all secondary centres.

- Children with septicemia had increased ESR and azotemia associated with thrombocytopenia.Since the number of septicemia cases was small,this association needs to be elaborated with larger studies.
- IgM dengue ELISA is sensitive in identifying dengue.Along with NS1 and IgG,it is useful in categorizing primary and secondary infections and thus identify predisposition to severe dengue.
- Transfusion of blood products have not significantly altered the outcome to a great extent.Also most thrombocytopenia in the present study was transient and the counts recovered in due time with treatment of the primary pathology.Therefore,guidelines need to be followed while transfusing.It is prudent to treat the clinical presentation in the patient rather than treat the platelet numbers.There is no role for prophylactic transfusions,as platelet counts do not correlate with bleeding.
- Awareness on dengue is poor among the rural population.People have to be sensitized about the signs and symptoms of the disease and the importance of mosquito eradication.Awareness campaigns need to be stepped up.
- As thrombocytopenia is so common and has a definite bearing on prognosis in the intensive care setting,it needs to be studied further.With the paucity of studies worldwide on paediatric thrombocytopenia, studies are required with larger number of patients in

the paediatric age group to further consolidate the findings of the present study.

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APPENDIX

1. PROFORMA FOR THROMBOCYTOPENIA

NAME: IP NO: AGE/SEX:

ADDRESS: MOBILE NO:

DOA: ADMISSION WT:

DOD: DISCHARGE WT:

FATHERS NAME: MOTHERS NAME:

OCCUPATION: INCOME:

CONSANGUINITY: SIBLINGS:

FAMILY HISTORY;

ILLNESS STARTED ON: DAY OF PRES:

PRE HOSPITAL TREATMENT:DRUGS:

IV FLUIDS:

GENERAL CONDITION:

TRANSFUSIONS:

DIAGNOSIS:

DAY OF ILLNESS PRES:

PVT/GOVT SET UP:

CHIEF PRESENTING COMPLAINT WITH DURATION:

FEVER : TYPE: PERIODICITY:

CHILLS/RIGORS: SWEATING EPISODES:

RELIEF WITH ANTIPYRETICS:

COUGH:

VOMITTING:

HEMATEMESIS:

MALENA:

RASH:

HEMATURIA:

LOOSE STOOLS:

ABD PAIN:

ALOC/SEIZURES:

BONE PAINS:

BREATHLESSNESS:

REFUSAL OF FEEDS:

LOW/LOA:

IRRITABLE CRY:

FEATURES OF EDEMA:

ANTECEDENT ILLNESS IN THE PAST MONTH:

OTHERS:(OTHER COMPLAINTS/COMORBIDITIES)

DRUG INTAKE/DAYS:

PAST HISTORY:

H/O BLOOD TRANSFUSION:

DEV HISTORY: IMM STATUS:

GENERAL EXAMINATION AT PRES:

GC		AVPU		TACHYPNEIC	
TEMP		HR		RR	
PULSES		PALLOR		ICTERUS	
LYMPH NODES		BP		TOURNIQUET TEST	
FACIAL PUFFINESS		PEDAL EDEMA		CYANOSIS	
PERIPHERIES		WEIGHT		WT PERCENT	
HEIGHT		HT PERCENT		AIRWAY	
SP O2		PUPILS		FUNDUS	
COLOUR		GCS		BONE TENDERNESS	

OTHER FINIDNGS IF ANY:

CVS:

RS:

P/A(LIVER SPAN):

CNS:

DESCRIPTION OF RASH:

PALPABLE/NON PALPABLE: SIZE:

COLOUR: BLANCHING/REFILL:

INVESTIGATIONS:

INV	DATE	REPORT	DATE	REPORT	DATE	REPORT	DATE	REPORT
TC								
DC								
ESR								
HB								
PCV								
PT COUNT								
BLEEDING MANIFESTATION								
SUGAR								
AMYLASE								
UREA								
CREATININE								
NA								
POT								
BILIRUBIN								
SGOT								
SGPT								
ALP								
ALBUMIN								
GLOBULIN								
A/G RATIO								
URINE A S D								

URINE C/S:

BLOOD C/S:

IgM DENGUE ELISA:

PERIPHERAL SMEAR

RETIC COUNT:

WIDAL:

BT; CT:

PT:

APTT:

HIV ELISA:

ANA:

CXR:

USG ABDOMEN:

BONE MARROW:

OTHERS IF ANY:

INITIAL TREATMENT:

IVF: ABC: INOTROPES:

COURSE OF ILLNESS:

TRANSFUSIONS WITH DATE: BLD GP:

PRODUCT	DT			

STEROIDS: DATE STARTED:

DATE													
FLUID													
INPUT													
OUTPUT													
TEMP													

SPIKE:

COMPLICATIONS:

FINAL DIAGNOSIS:

FOLLOW UP GC

PLATELET LEVEL:

REMARKS:

QUESTIONNAIRE:

1.HAVE YOU HEARD THE WORD “DENGUE”?

2.TELL THE SIGNS AND SYMPTOMS OF THE DISEASE

3.HOW IS THE DISEASE TRANSMITTED?

2. ABBREVIATIONS USED IN THE TEXT

- VWF-VON WILLEBRAND FACTOR
- SLE-SYSTEMIC LUPUS ERYTHEMATOSUS
- ALL-ACUTE LYMHOBLASTIC LEUKEMIA
- PT-PROTHROMBIN TIME
- APTT –ACTIVATED PARTIAL THROMBOPLASTIN TIME
- DENV-DENGUE VIRUS
- HCT-HAEMATOCRIT
- ADP-ADENOSINE DIPHOSPHATE
- ATP-ADENOSINE TRIPHOSPHATE
- HIV-HUMAN IMMUNODEFICIENCY VIRUS
- ITP-IDIOPATHIC THROMBOCYTOPENIC PURPURA
- EBV-EBSTEIN BARR VIRUS
- CMV-CYTOMEGALOVIRUS
- HCV- HEPATITIS C VIRUS
- HIT-HEPARIN INDUCED THROMBOCYTOPENIA
- TTP-THROMBOTIC THROMBOCYTOPENIC PURPURA
- HUS-HEMOLYTIC UREMIC SYNDROME
- NS-NON STRUCTURAL PROTEIN
- EDTA-ETHYLENE DI AMINE TETRA ACETIC ACID
- WHO-WORLD HEALTH ORGANISATION
- DF-DENGUE FEVER
- DHF-DENGUE HEMORRHAGIC FEVER
- DIVC-DISSEMINATED INTRAVASCULAR COAGULATION
- PUO-PYREXIA OF UNKNOWN ORIGIN
- ICU-INTENSIVE CARE UNIT
- ESR-ERYTHROCYTE SEDIMENTATION RATE

- ICB/H-INTACRANIAL BLEED/HEMORRHAGE
- MPV-MEAN PLATELET VOLUME
- RBC-RED BLOOD CELL
- DSS-DENGUE SHOCK SYNDROME
- SD-STANDARD DEVIATION

MASTER CHART

S.NO	IP NO	AGE	SEX	WT	WT FOR AGE	PRE HOSP	ADEQ	STAY	ADM FEVER	TOTAL FEVER	COUGH	VOMITING
1	59151	9	F	20	II	PVT	I	3	4	10	N	Y
2	58928	5	M	14	I	GH	A	5	4	4	Y	Y
3	59387	3	M	10	I	GH	A	7	7	12	Y	Y
4	60598	3	M	12	N	SELF	NA	4	6	7	N	Y
5	59915	8	F	20	N	GH	I	7	3	7	Y	Y
6	59983	2	M	10	N	PVT	A	6	2	5	N	N
7	60194	7	M	20	N	PVT	NA	8	4	5	Y	Y
8	60318	12	M	23	III	PVT	A	5	5	7	Y	N
9	61450	11	M	35	N	GH	A	5	8	9	N	Y
10	61188	2	F	6.5	III	GH	A	3	7	8	N	Y
11	61225	7	M	14	II	SELF	NA	5	7	5	Y	Y
12	61183	12	F	24	III	SELF	NA	12	4	5	N	N
13	232	8	F	22	N	SELF	NA	7	3	5	N	Y
14	384	1	F	8.5	N	GH	A	7	10	10	Y	N
15	507	5	M	18	N	PVT	A	6	7	7	Y	Y
16	681	10	M	30	N	PVT	I	4	7	7	N	Y
17	509	3	F	12	N	PVT	A	4	4	5	N	Y
18	944	7	F	17	I	GH	A	4	7	9	N	Y
19	1384	10	M	23	I	GH	A	6	4	4	Y	Y
20	1651	9	M	20	II	PVT	A	4	7	7	N	N
21	1428	5	M	12	II	PVT	I	5	5	5	N	N
22	1419	9	F	15	III	PVT	I	5	4	4	N	N
23	1459	4	M	11	I	PVT	A	6	5	9	Y	N
24	1489	8	M	25	N	SELF	NA	4	2	3	N	Y
25	1913	7	F	19	N	GH	A	4	2	6	Y	Y
26	1904	10	F	22	I	GH	A	5	5	6	N	Y
27	1916	2	M	12	N	GH	A	4	6	8	N	N
28	2782	9	M	20	II	PVT	A	7	2	4	Y	N

S.NO	IP NO	AGE	SEX	WT	WT FOR AGE	PRE HOSP	ADEQ	STAY	ADM FEVER	TOTAL FEVER	COUGH	VOMITING
29	3967	7	F	16	I	GH	A	4	5	5	N	N
30	3574	7	F	20	N	PVT	I	5	7	10	Y	Y
31	4087	3	M	10	I	PVT	I	4	7	8	N	N
32	3687	5	M	13	I	GH	A	4	5	5	N	Y
33	3822	9	F	20	II	PHC	I	6	7	9	N	Y
34	3842	10	M	30	N	GH	A	6	7	8	N	N
35	3829	2	M	8	II	SELF	NA	7	5	8	N	N
36	4525	10	M	24	I	PVT	A	3	5	5	N	N
37	4265	5	F	14	I	GH	I	4	3	3	N	Y
38	4714	10	M	20	II	GH	I	5	3	6	Y	Y
39	4740	8	M	18	I	PVT	A	5	7	7	N	Y
40	5201	7	F	14	II	PHC	A	4	4	5	Y	Y
41	4882	12	M	23	III	SELF	NA	8	4	6	Y	Y
42	5228	8	F	17	I	PVT	A	2	2	4	Y	Y
43	5575	8	F	18	I	PVT	A	4	2	2	N	N
44	6109	4	M	18	N	PVT	A	5	3	4	N	N
45	6557	4	M	12	II	SELF	NA	11	7	9	Y	Y
46	6454	8	M	18	I	GH	A	5	5	6	N	N
47	6462	9	M	25	N	SELF	NA	4	4	6	N	N
48	6411	10	M	25	I	GH	A	10	3	8	N	Y
49	2767	9m	F	8	N	PVT	A	12	4	10	N	Y
50	4246	6	F	15	I	SELF	NA	3	5	5	N	Y
51	2377	6m	F	5.5	N	PVT	A	8	3	6	Y	Y
52	588	3	F	12	N	PVT	A	8	6	6	Y	Y
53	1646	9	F	16.5	III	PVT	I	4	5	5	Y	Y
54	60099	12	F	24	III	GH	I	4	5	5	N	N
55	960	8	M	18	I	GH	I	5	1	4	N	Y
56	4053	12	F	32	N	GH	I	4	4	5	N	Y

S.NO	IP NO	AGE	SEX	WT	WT FOR AGE	PRE HOSP	ADEQ	STAY	ADM FEVER	TOTAL FEVER	COUGH	VOMITING
57	5075	3	M	12	N	PVT	A	10	4	10	Y	Y
58	3233	7	F	18	N	PVT	A	3	7	7	N	Y
59	5186	9	M	21	II	GH	I	5	5	6	N	N
60	4673	5	M	15	I	GH	A	8	3	7	N	Y
61	179	8	M	21	N	GH	A	8	7	9	Y	Y
62	60277	12	M	25	II	PVT	A	4	4	4	N	N
63	5790	8m	F	7	N	PVT	A	1	5	6	N	N
64	1290	2	F	10	N	PVT	A	2	3	5	N	N
65	60813	1	M	8	N	PVT	A	7	5	7	N	Y
66	1927	6	F	18	N	PVT	A	4	15	18	Y	Y
67	6690	7	M	17	I	SELF	NA	12	3	8	Y	Y
68	60157	3	F	11	N	PVT	I	5	5	7	N	Y
69	59921	8	F	16	II	SELF	NA	6	3	8	N	Y
70	588	3	F	12	N	PVT	A	6	6	6	N	Y
71	869	4	M	16	N	GH	A	5	7	10	N	Y
72	1785	12	M	25	III	GH	A	9	5	5	Y	N
73	2063	4	F	12	II	PVT	A	5	5	6	N	Y
74	5634	7	M	23	N	SELF	NA	6	5	5	Y	Y
75	876	11	F	28	I	SELF	NA	9	3	12	N	Y
76	2672	11	M	24	II	PHC	I	5	10	12	N	Y
77	4105	7	F	24	N	PVT	A	5	5	7	N	Y
78	59494	5	M	16	N	SELF	NA	4	15	16	Y	Y
79	5442	11	F	28	I	PVT	I	7	9	12	N	N
80	5106	10	F	25	I	PVT	I	5	15	17	N	Y
81	5807	10	M	20	II	SELF	NA	15	5	12	Y	Y
82	60712	2	F	10	N	PVT	I	4	15	15	Y	Y
83	60446	6	M	10	IV	SELF	NA	8	15	20	N	N
84	59383	7m	M	6	N	GH	NA	7	10	14	Y	Y

S.NO	IP NO	AGE	SEX	WT	WT FOR AGE	PRE HOSP	ADEQ	STAY	ADM FEVER	TOTAL FEVER	COUGH	VOMITING
85	59413	1	M	6	II	GH	A	25	7	15	Y	Y
86	682	10m	F	7.3	N	PVT	I	6	5	5	Y	N
87	2011	2m	F	3	N	SELF	NA	2	4	6	Y	Y
88	72	2m	F	3.5	N	GH	A	2	5	6	Y	Y
89	59811	8	F	14	III	PVT	A	5	5	7	N	Y
90	4700	7	F	15	II	GH	A	4	5	6	N	Y
91	6821	5	F	18	N	PVT	A	8	4	6	Y	N
92	60900	12	F	25	II	PVT	A	8	10	11	N	Y
93	1490	6	F	12	II	SELF	NA	2	14	14	N	N
94	4163	5	F	13	I	PVT	A	7	7	8	N	Y
95	14369	10	F	26	N	SELF	NA	2	10	12	Y	N
96	20573	9	F	16.5	III	SELF	NA	8	8	15	N	N
97	6965	3	F	13	N	GH	A	10	AFEB	AFEB	N	N
98	15117	10	F	18	III	SELF	NA	10	AFEB	AFEB	Y	N
99	22862	10	F	27	N	SELF	NA	1	AFEB	AFEB	N	Y
100	13649	12	M	28	I	PVT	A	8	AFEB	AFEB	N	Y
101	4745	8	F	17	II	PVT	I	5	4	4	Y	Y
102	5047	5m	M	5	N	PVT	A	2	10	12	N	N
103	296	6	F	13	II	SELF	NA	3	5	6	N	N
104	576	5	M	13	I	PVT	I	20	2	15	N	Y
105	4877	12	F	20	III	SELF	NA	6	4	6	Y	N
106	4622	7	M	23	N	SELF	NA	15	10	10	Y	N
107	5786	3	F	10	N	SELF	NA	9	AFEB	AFEB	N	N
108	2271	9	F	25	N	PVT	I	6	5	6	Y	Y
109	59752	12	M	26	II	SELF	NA	12	2	7	N	Y
110	61457	5	F	15	N	GH	A	9	4	8	N	Y
111	2244	6	F	15	I	GH	A	7	7	8	Y	Y
112	5588	2	M	15	N	GH	A	6	4	6	N	Y

S.NO	MYALGIA	ABD PAIN	TEND	DIST	OLIGURIA	PUFF FACE	PEDAL EDEMA	HEMATEMESIS	MALENA	BLEEDING	HESS TEST
1	Y	Y	Y	N	N	N	N	N	N	N	N
2	N	Y	N	N	N	P	N	N	Y	GI	P
3	Y	N	N	N	Y	N	N	N	N	N	N
4	N	N	N	N	N	N	N	N	N	PURPURA	P
5	Y	N	N	N	N	P	N	N	N	N	N
6	Y	N	N	N	N	N	N	N	N	N	N
7	Y	N	N	Y	N	N	N	N	N	PETECHIAE	N
8	N	Y	N	N	N	N	N	N	N	N	N
9	Y	Y	N	N	N	N	N	N	Y	GI	N
10	N	Y	N	Y	N	P	N	N	Y	GI	N
11	Y	N	N	N	Y	P	N	N	N	N	N
12	Y	N	N	N	N	N	N	N	Y	GI	N
13	N	N	N	N	N	N	N	N	N	N	N
14	Y	N	N	Y	N	N	N	N	N	N	N
15	Y	Y	N	Y	Y	P	N	N	N	N	N
16	N	Y	Y	Y	N	P	P	N	Y	GI	P
17	N	Y	N	Y	N	P	N	N	N	PETECHIAE	N
18	N	N	N	N	N	P	N	N	N	N	N
19	Y	Y	N	N	N	N	N	N	Y	GI	P
20	Y	Y	N	N	N	N	N	N	N	N	N
21	Y	Y	Y	N	N	N	N	N	N	N	N
22	Y	Y	Y	N	N	P	N	N	N	N	P
23	N	Y	Y	N	Y	P	N	N	N	N	N
24	Y	Y	Y	N	N	N	N	N	N	N	N
25	N	N	N	N	N	N	N	N	N	N	N
26	Y	Y	N	N	N	N	N	N	Y	GI	N
27	Y	Y	N	N	N	N	N	N	Y	GI	N
28	N	Y	Y	N	N	N	N	N	N	PETECHIAE	P

S.NO	MYALGIA	ABD PAIN	TEND	DIST	OLIGURIA	PUFF FACE	PEDAL EDEMA	HEMATEMESIS	MALENA	BLEEDING	HESS TEST
29	Y	N	N	N	N	N	N	N	N	N	N
30	Y	Y	N	N	Y	N	N	Y	Y	GI	N
31	Y	N	N	N	N	N	N	N	Y	GI	P
32	N	N	N	N	N	N	N	Y	N	GI	N
33	N	Y	N	N	N	N	N	N	Y	PETECHIAE	N
34	Y	Y	Y	Y	N	N	N	N	N	N	P
35	Y	N	N	N	N	N	N	N	N	N	N
36	N	N	N	N	N	N	N	N	N	N	N
37	N	Y	N	N	N	N	N	Y	N	GI	N
38	Y	Y	N	N	N	N	N	N	N	EPISTAXIS	N
39	N	Y	Y	N	N	N	N	N	Y	GI	P
40	Y	Y	N	N	N	N	N	N	Y	GI	N
41	Y	Y	N	Y	N	N	N	Y	N	GI	P
42	N	Y	N	N	N	N	N	Y	N	GI	N
43	N	N	N	N	N	N	N	N	N	N	N
44	Y	Y	N	N	N	N	N	N	N	N	N
45	N	Y	Y	N	N	N	N	N	N	N	N
46	Y	Y	N	N	N	N	N	N	Y	GI	N
47	N	N	N	N	N	N	N	N	N	N	N
48	Y	Y	N	N	N	N	N	Y	Y	GI	P
49	Y	N	N	Y	N	P	N	Y	Y	GI	N
50	N	N	N	N	N	N	N	N	N	N	N
51	N	N	N	Y	N	P	P	N	Y	GI	P
52	N	Y	Y	Y	N	P	P	N	Y	GI	N
53	Y	N	N	N	N	N	N	Y	N	GUM BLEED	N
54	Y	N	N	N	N	N	N	N	N	N	N
55	N	N	N	N	N	N	N	N	N	N	N
56	N	Y	N	N	N	N	N	N	N	N	N

S.NO	MYALGIA	ABD PAIN	TEND	DIST	OLIGURIA	PUFF FACE	PEDAL EDEMA	HEMATEMESIS	MALENA	BLEEDING	HESS TEST
57	Y	N	N	N	N	P	P	N	Y	GI	N
58	N	Y	N	Y	N	N	N	N	Y	PURPURA	P
59	Y	N	N	N	N	N	N	N	N	N	N
60	N	Y	Y	Y	N	N	N	N	Y	GI	P
61	N	N	N	N	Y	N	N	N	Y	GI	N
62	Y	N	N	N	N	N	N	N	Y	GI	N
63	N	N	N	Y	N	N	N	Y	Y	IVSITE/SC ECHYMOSIS	P
64	N	N	N	Y	N	P	P	Y	Y	GI	N
65	N	N	N	Y	N	P	P	N	Y	GI	P
66	N	N	N	N	Y	N	N	N	N	N	N
67	Y	Y	N	N	N	N	N	Y	Y	GI	P
68	Y	N	N	N	N	P	N	Y	Y	GI	N
69	N	N	N	N	N	N	N	N	N	EPISTAXIS	N
70	Y	Y	Y	Y	N	P	P	N	Y	GI	N
71	Y	Y	N	N	Y	N	N	Y	Y	GI	N
72	N	Y	N	N	N	N	N	N	N	N	N
73	N	Y	N	N	N	N	N	N	N	N	N
74	Y	Y	N	N	Y	N	N	Y	Y	GI	N
75	N	Y	N	N	N	N	N	N	N	N	N
76	N	Y	Y	Y	N	N	N	N	Y	GI	N
77	Y	N	N	N	N	N	N	N	Y	GI	N
78	N	N	N	N	N	N	N	N	N	N	N
79	Y	Y	N	N	N	N	N	N	N	N	N
80	N	Y	Y	Y	N	N	N	N	N	PURPURA	N
81	Y	Y	Y	N	N	N	N	N	N	PETECHIAE	N
82	N	Y	N	N	N	N	N	Y	Y	PURPURA	N
83	Y	Y	N	Y	N	N	N	N	N	N	P
84	N	N	N	Y	Y	P	N	N	N	N	N

S.NO	MYALGIA	ABD PAIN	TEND	DIST	OLIGURIA	PUFF FACE	PEDAL EDEMA	HEMATEMESIS	MALENA	BLEEDING	HESS TEST
85	N	N	N	N	N	N	N	N	Y	GI	N
86	N	N	N	N	N	P	N	N	N	PETECHIAE	N
87	N	N	N	N	Y	N	N	N	N	N	N
88	N	N	N	Y	Y	N	N	Y	N	GI	N
89	N	N	N	N	N	P	P	N	Y	GI	N
90	Y	Y	N	N	N	N	N	N	N	N	N
91	N	N	N	N	N	P	N	N	N	N	N
92	N	N	N	N	N	N	N	N	N	PETECHIAE	N
93	Y	N	N	N	N	N	N	N	N	N	N
94	Y	Y	N	N	N	N	P	N	N	N	N
95	N	N	N	N	N	N	N	N	Y	PETECHIAE	N
96	Y	N	N	N	N	N	N	N	N	PETECHIAE	N
97	Y	N	N	N	N	N	N	N	N	PURPURA/ECCHYMOSIS	N
98	N	N	N	N	N	N	N	N	Y	PURPURA/GUM BLEED	N
99	N	Y	N	Y	N	P	P	Y	Y	PETECHIAE	N
100	Y	Y	N	Y	Y	P	N	Y	Y	GI	N
101	Y	Y	N	N	N	N	N	N	N	N	N
102	N	N	N	Y	N	N	N	Y	N	GI	N
103	Y	Y	N	N	N	N	N	N	N	N	N
104	N	Y	Y	N	N	P	N	N	Y	GI	N
105	Y	Y	N	N	N	N	N	N	N	GUM/PETECHIAE	N
106	N	N	N	N	N	N	N	N	N	EPISTAXIS	N
107	Y	Y	N	Y	N	N	N	N	N	N	N
108	Y	Y	N	Y	Y	N	N	N	Y	GI	N
109	N	Y	Y	N	N	N	N	Y	Y	GI	N
110	Y	Y	N	Y	Y	P	P	Y	Y	GI	N
111	N	N	N	N	N	P	N	Y	N	GI	N
112	Y	N	N	N	N	N	N	Y	N	GI	P

S.NO	RASH	AVPU	TACHYCARDIA	TACHYPNEA	PULSE PRES	SHOCK	SEIZURES	INOTROPE	VENTILATION	TC	HB	ESR	REF CNT
1	ERYTH	A	N	N	30	N	N	N	N	4300	14	N	85000
2	N	A	N	N	30	N	N	N	N	7900	11.9	I	
3	ERYTH	IRRITABLE	N	N	20	N	N	N	N	12400	8.1	N	110000
4	N	A	Y	N	30	N	Y	N	N	15400	10.1	N	
5	FLUSH	DROWSY	N	N	30	N	N	N	N	5,400	11	N	
6	ERYTH	A	N	N	25	N	N	N	N	6200	11.9	N	2,66,000
7	ERYTH	LETHARGIC	N	N	25	N	N	N	N	4100	12	I	2,50,000
8	FLUSH	A	N	N	40	N	N	N	N	2700	13.7	N	99000
9	N	A	N	N	30	N	N	N	N	6300	12.9	N	58000
10	ERYTH	LETHARGIC	Y	D	30	N	N	N	N	12600	12.1	N	49000
11	N	LETHARGIC	N	D	25	N	N	N	N	5100	13.2	N	
12	OLD HEALED	LETHARGIC	Y	N	25	N	N	N	N	2000	3.3	I	
13	FLUSH	A	N	N	25	N	N	N	N	3400	12.2	N	
14	ERYTH	IRRITABLE	N	D	30	N	N	N	N	13800	10.7	N	75000
15	N	A	Y	N	25	N	N	N	N	2700	12.2	N	
16	ERYTH	TOXIC	N	N	30	N	N	N	N	14000	14.1	I	
17	ERYTH	A	N	N	20	N	N	N	N	4400	12	N	99000
18	ERYTH	A	Y	N	30	N	N	N	N	5000	13.6	N	57000
19	FLUSH	A	Y	N	30	N	N	N	N	3400	12.5	N	
20	N	A	Y	N	20	N	N	N	N	5200	14.9	N	450000
21	ERYTH	A	Y	N	30	N	N	N	N	9300	14.5	I	
22	N	A	Y	N	35	N	N	N	N	4500	14.5	N	
23	ERYTH	A	N	N	40	N	N	N	N	3800	11.2	N	93000
24	N	LETHARGIC	N	N	30	N	N	N	N	10400	12	N	
25	MACULOPAP	A	N	N	25	N	N	N	N	4400	9.5	N	47000
26	N	A	N	N	25	N	N	N	N	10200	10.9	N	60000
27	ERYTH	A	N	N	30	N	N	N	N	10900	9.7	N	73000
28	ERYTH	IRRITABLE	N	N	40	N	N	N	N	7400	12.4	N	625000

S.NO	RASH	AVPU	TACHYCARDIA	TACHYPNEA	PULSE PRES	SHOCK	SEIZURES	INOTROPE	VENTILATION	TC	HB	ESR	REF CNT
29	N	A	Y	N	25	N	N	N	N	3000	10.9	N	79000
30	ERYTH	IRRITABLE	Y	N	30	N	N	N	N	1800	12.5	N	
31	N	DROWSY	N	N	30	N	N	N	N	9100	10.2	N	
32	N	A	N	N	30	N	N	N	N	18000	12.9	I	59000
33	ERYTH	TOXIC	N	N	30	N	N	N	N	7800	15.2	N	
34	FLUSH	LETHARGIC	Y	N	30	Y	N	N	N	4100	13.1	N	72000
35	N	A	N	N	25	N	N	N	N	2700	10.3	N	
36	N	A	N	N	30	N	N	N	N	2700	11.5	I	170000
37	FLUSH	A	Y	N	25	N	N	N	N	6500	12.2	N	
38	FLUSH	A	N	N	30	N	N	N	N	10300	12.6	I	100000
39	N	LETHARGIC	N	N	20	N	N	N	N	14100	10.5	N	
40	N	SICK LOOK	Y	N	30	N	N	N	N	5400	13.1	N	25000
41	FLUSH	A	Y	N	20	N	N	N	N	5000	13.1	N	
42	ERYTH/FLUSH	DROWSY	Y	D	30	Y	N	Y	N	5700	12.8	N	
43	N	A	N	N	20	N	N	N	N	13400	11.6	I	52000
44	N	A	Y	N	30	N	N	N	N	4000	13.4	N	
45	ERYTH	A	N	N	40	N	N	N	N	6000	14.5	N	
46	N	A	N	N	30	N	N	N	N	13000	11.3	N	90000
47	ERYTH	A	N	N	40	N	N	N	N	4100	11.7	N	
48	FLUSH	A	N	N	30	N	N	N	N	3000	11.7	I	169000
49	FLUSH	IRRITABLE	Y	D	20	N	N	N	N	18800	10.8	N	44000
50	N	A	Y	N	25	N	N	N	N	6500	12.8	N	
51	ERYTH	LETHARGIC	Y	D	30	Y	N	N	N	12000	11.2	N	43000
52	N	A	Y	N	30	N	N	N	N	11800	11.9	N	68000
53	ERYTH	A	N	N	20	N	N	N	N	5800	12.2	I	
54	N	A	N	N	30	N	N	N	N	3400	13	N	
55	N	A	N	N	30	N	N	N	N	8300	12	I	
56	ERYTH	A	N	N	40	N	N	N	N	5800	11.2	N	

S.NO	RASH	AVPU	TACHYCARDIA	TACHYPNEA	PULSE PRES	SHOCK	SEIZURES	INOTROPE	VENTILATION	TC	HB	ESR	REF CNT
57	ERYTH	A	N	N	20	N	N	N	N	2600	7.9	N	
58	N	LETHARGIC	Y	N	30	Y	Y	N	N	10900	11.4	N	33000
59	ERYTH	A	N	N	20	N	N	N	N	3500	12.8	I	
60	N	DROWSY	N	N	30	N	Y	N	N	6000	11.3	I	369000
61	FLUSH	TOXIC	N	N	30	N	N	N	N	4600	11.6	I	
62	N	A	N	N	25	N	N	N	N	8400	14.5	N	
63	ERYTH	DROWSY	Y	D	30	Y	N	N	N	9800	5.7	N	
64	ERYTH	STATUS	Y	D	30	Y	Y	Y	Y	14100	8.8	N	
65	ERYTH	IRRITABLE	Y	D	20	Y	N	Y	Y	3800	11.2	N	38000
66	ERYTH	DROWSY	Y	N	30	Y	N	N	N	9200	15.2	N	69000
67	N	DROWSY	Y	N	80	Y	N	N	N	3500	11.5	N	254000
68	N	A	N	N	25	N	N	N	N	12800	13	I	90000
69	N	DROWSY	N	N	30	N	N	N	N	8000	12.6	N	
70	N	A	Y	N	30	N	N	N	N	11800	11.9	I	68000
71	ERYTH	A	N	N	25	N	N	N	N	8200	13.2	I	78000
72	N	A	Y	N	40	N	N	N	N	2800	10.3	I	
73	N	A	N	N	30	N	N	N	N	3100	12.7	N	110000
74	N	DROWSY	Y	N	30	Y	N	N	N	4000	14	N	
75	ERYTH	DROWSY	N	N	30	N	N	N	N	18000	13.2	N	
76	N	TOXIC	Y	N	30	N	N	N	N	4100	12	N	
77	ERYTH	A	N	N	25	N	N	N	N	3500	12.9	I	73000
78	ERYTH	A	Y	N	30	N	N	N	N	11100	10	N	368000
79	N	A	N	N	25	N	N	N	N	4600	9.9	I	
80	ERYTH	DROWSY	Y	D	30	Y	Y	N	N	3900	12.3	I	
81	FLUSH	A	N	N	25	N	N	N	N	3800	12.8	I	
82	ERYTH	IRRITABLE	Y	N	20	N	N	N	N	11000	12.3	N	2,20,000
83	DRY ?ADE	TOXIC	N	N	30	N	N	N	N	17700	8.8	I	
84	N	A	N	D	30	N	Y	Y	Y	5000	11.8	I	195000

S.NO	RASH	AVPU	TACHYCARDIA	TACHYPNEA	PULSE PRES	SHOCK	SEIZURES	INOTROPE	VENTILATION	TC	HB	ESR	REF CNT
85	N	IRRITABLE	N	N	25	N	Y	N	N	5200	9.4	I	
86	ERYTH	A	N	N	30	N	N	N	N	8500	10.9	I	
87	N	DROWSY	Y	D	20	Y	Y	Y	Y	3900	8.7	I	
88	N	DROWSY	Y	D	25	Y	Y	Y	Y	11000	9	N	
89	ERYTH	LETHARGIC	N	N	36	N	N	N	N	17200	7.3	I	41000
90	N	LETHARGIC	N	N	30	N	N	N	N	3200	12.4	N	72000
91	N	A	Y	N	20	N	N	N	N	5100	13.4	I	
92	ERYTH	LETHARGIC	N	N	30	N	N	N	N	1100	6.6	I	
93	N	A	N	N	30	N	N	N	N	35300	1.8	I	
94	N	A	Y	N	30	N	N	N	N	25600	2.7	I	
95	N	A	N	N	30	N	N	N	N	3300	4.5	N	
96	N	A	N	N	25	N	N	N	N	32800	5.7	I	
97	ERYTH	A	N	N	25	N	N	N	N	11600	9.6	N	16000
98	N	A	N	N	25	N	N	N	N	5000	9.2	I	
99	N	DROWSY	Y	D	30	Y	N	Y	Y	5600	8.3	I	
100	N	DROWSY	Y	D	30	N	N	N	N	12000	8.7	N	
101	ERYTH	A	N	N	30	N	N	N	N	7300	12.5	N	
102	ERYTH	LETHARGIC	Y	D	25	Y	N	Y	Y	3000	5	I	
103	N	A	N	N	30	N	N	N	N	3800	8.1	N	
104	ERYTH	DROWSY	Y	N	20	N	N	N	N	15400	9.1	I	
105	N	A	Y	N	30	N	N	N	N	2900	7	N	
106	N	A	N	N	20	N	N	N	N	6700	11.8	N	
107	N	A	N	N	30	N	N	N	N	2400	5.7	N	
108	ERYTH	TOXIC	Y	D	20	Y	N	N	N	3300	15.9	N	
109	N	IRRITABLE	Y	D	34	N	Y	N	N	15,700	15.9	I	
110	FLUSH	P	Y	N	20	N	Y	N	N	22700	9.5	I	
111	N	P	Y	D	20	Y	Y	N	N	22500	8.7	N	
112	N	U	Y	D	50	Y	Y	N	N	33300	12.4	I	

S.NO	R 1	PCV	R2	PCV2	R3	PCV3	R4	R5	UREA	CREAT	SUGAR	LIVER ENZ	BILIRUBIN	IGM	IGG	NSI	WIDAL
1	80,000	42	72000	42.6	120000	43.5	200000		18	0.8	78	N	N	P	P	P	N
2	74000	37.4	90000	36	120000	36			29	0.8	60	N	N	P	N	N	N
3	58000	30.2	1,10,000	31.8	175000	27.9			48	0.8	60	N	N	P	P	N	N
4	47000	31.8	1,29,000						68	1	21	I 200	I	P	P	N	N
5	1,49,000	34.4	25,000	37	23,000	40	15,000	1,29,000	32	1	54	N	N	P	P	N	N
6	2,02,000	36.7	99,000	37.3	150000	36			30	0.9	67	N	N	N	N	P	N
7	1,80,000	38.7	39,000	37	90000	38			55	1.5	148	N	N	P	N	N	N
8	96000	43.2	1,00,000	36	2,72,000	35			25	0.9	38	N	N	N	P	N	N
9	79000	40.8	211,000	42					16	0.6	139	N	N	P	P	N	N
10	47000	39.1	72,000	39	142000	37			18	0.8	73	N	N	P	P	N	N
11	25000	41.7	37000	34.5	72000	34	145000		22	0.7	75	N	N	P	N	P	N
12	29000	10.1	41000	17.4	94000	18	102000		15	0.7	104	N	N	P	N	N	N
13	119,000	39.3	79000	39.4	120000	38			20	0.8	87	N	N	N	P	N	N
14	79000	34.1	135000	34.5					25	0.8	60	N	N	P	N	P	N
15	84000	36.7	94000	37.9	145000	37.3			18	0.8	65	I 300	N	P	P	N	N
16	27000	40	40000	41	67000	40	122000		41	0.9	76	I 200	N	P	P	N	N
17	92000	38.6	144000	36					19	0.7	80	N	N	P	P	P	N
18	46000	39.9	54000	40.2	76000	40	137000		19	0.9	74	I 3000	N	P	P	N	N
19	60000	37	15000	39	82000	34.1	150000		24	36	1.4	N	N	P	P	N	N
20	68000	45.5	90000	38	200000	36			19	0.9	65	I 200	N	P	P	P	N
21	46000	33	25000	35	58000	37	72000	175000	42	1	60	I 200	N	P	P	N	N
22	21000	46.2	43000	37	79000	36.8	120000		46	1.5	107	N	N	P	P	N	N
23	87000	38.9	131000	35	225000	36.3			18	0.7	82	I 300	N	P	P	N	N
24	92000	37.7	100000	36	250000	37			23	0.8	89	N	N	P	N	P	N
25	43000	37.1	145000	30.1	140000	32.6			18	0.8	85	N	N	P	P	N	N
26	62000	32.8	97000	33	167000	34			35	1.1	35	N	N	P	N	N	N
27	90000	33.1	125000	34					18	0.8	8	N	N	P	N	P	N
28	46000	54.2	25000	53.3	21000	45.5	33000	102000	37	1.2	92	I 519	N	P	N	N	N

S.NO	R 1	PCV	R2	PCV2	R3	PCV3	R4	R5	UREA	CREAT	SUGAR	LIVER ENZ	BILIRUBIN	IGM	IGG	NSI	WIDAL
29	84000	35	120000	36					29	0.8	95	N	N	N	N	P	N
30	103000	38.2	82000	38.9	127800	37			18	0.8	62	N	N	P	N	N	N
31	78000	37.5	141000	36					33	1.2	72	N	N	P	P	P	N
32	55000	43	238000	40					21	0.8		N	N	P	P	N	N
33	20000	47.4	31000	45	54000	43	79000	135000	45	1.3	57	N	N	N	N	P	N
34	52000	49.6	26000	41	36000	41.2	98000	123000	26	1	50	N	N	P	N	N	N
35	79000	34.6	98000	33.9	129000	32			26	1	76	N	N	P	N	P	N
36	47000	37.8	74000	36.7	147000	35			45	0.8	118	N	N	N	N	P	N
37	33000	39.4	118000	33.5					45	1.3	75	N	N	P	N	P	N
38	23000	39.4	26000	39	100000	34	123000		45	1.3	62	N	N	P	N	N	N
39	18000	47.7	24000	39.4	74000	32	190500		34	1.1		I 1457	N	P	P	P	N
40	28000	55.9	19000	46.6	13000	39.2	47000	103000	49	1	109	I 518	N	P	P	N	N
41	45000	39.9	55000	38	76000	37.4	112000		41	1.3	113	I 488	N	P	P	P	N
42	42000	53.8	42000	52.5	12000	41.4	16000	18000	29	0.8	82	N	N	P	N	N	N
43	30000	38.2	47000	38.1	94000	37	123000		32	1.1	90	N	N	P	P	N	N
44	93000	42.2	118000	40					26	0.8	105	N	N	N	N	P	N
45	64000	46.3	57000	39.3	79000	38	145000		53	1.5	84	I 277	I	N	P	N	N
46	80000	33.9	227000	32					16	0.6		I 716	N	P	N	P	N
47	75000	36.2	123000	32.8					31	1.2	90	N	N	P	N	N	N
48	63000	36.6	20000	41.1	72000	40	134000		40	1		N	N	P	N	P	N
49	44000	36	49000	37	76000	35.2	133000		23	0.8	65	N	N	P	N	N	N
50	62000	34.4	86000	35	120000	33			26	1.1	106	N	N	N	P	N	N
51	43000	35.1	11000	29.6	16000	30	50000	72000	26	1.2		N	N	P	N	N	N
52	58000	36.7	97000	37.3	167000	36			34	1.1	101	I 400	N	P	N	P	N
53	65000	38	87000	37	112000	36.8			24	1	62	N	N	P	P	N	N
54	72000	39	98000	38	127000	35.7			31	1.2	94	N	N	N	P	N	N
55	76000	37.3	114000						24	0.8	46	N	N	P	N	P	N
56	64000	36.5	92000	35.5	158000	34			21	0.8	89	N	N	P	P	N	N

S.NO	R 1	PCV	R2	PCV2	R3	PCV3	R4	R5	UREA	CREAT	SUGAR	LIVER ENZ	BILIRUBIN	IGM	IGG	NSI	WIDAL
57	67000	25.5	125000	27.4					26	0.8	109	N	N	P	N	N	N
58	31000	34.7	36000	33	63000	32	67000	176000	22	1	62	N	N	P	N	N	N
59	42000	40.2	74000	41	89000	38	156000		26	0.8	117	I 208	N	P	N	N	N
60	104000	46.2	53000	34.1	76000	35.4	123000	150000	49	1.2	68	N	N	P	N	N	N
61	71000	35.7	86000	34	98000	33	125000		20	0.8	65	N	N	N	P	N	P
62	91000	44.4	102,000	36					32	0.8		N	N	P	P	P	N
63	37000	17.3	35000	17	30000	18	23000	7000	44	0.9		N	N	P	N	N	N
64	19000	30	39000	34.3	20000	32	18000	16000	42	1.3	22	N	N	P	N	N	N
65	30000	38.1	15,000	41.8	13000	32.1	11000	10000	30	1	90	N	N	P	P	N	N
66	45000	44	29000	45.3	33000	39.6	78000	124000	19	0.9	60	N	N	P	P	P	N
67	108000	39.8	59000	36.7	35000	44.1	20000	130000	49	1.2	90	N	N	P	N	N	N
68	76000	39	74,000	30	350000	31			56	1	65	N	I	N	N	N	P
69	2,79,000	40.5	60,000	42	74000	40	117000	200000	18	0.8	181	N	N	N	N	N	P
70	58000	36.7	97000	37.3	178000	36			34	1.1	101	I 400	N	N	N	N	P
71	68000	39.4	86000	38	145000	34			28	0.8	78	I 400	N	N	N	N	N
72	42000	39.1	44000	31.6	75000	32	88000	156000	23	0.8	68	I 650	N	N	N	N	P
73	67000	44.8	76000	43	110000	40	156000		24	0.8	79	I 650	N	N	N	N	P
74	77000	4.3	149000	36					25	0.7	84	N	N	N	N	N	P
75	43000	41.3	37000	34.5	48000	37.6	74000	122200	39	1.2	104	N	N	N	N	N	P
76	129000	37.5	79000	40.5	87000	40	136000		17	0.7	63	I 200	N	N	N	N	P
77	37000	41.4	39000	39.7	54000	38	131000		17	0.7	81	N	N	N	N	N	P
78	90000	31	96000	32	115000	32	178000		22	1	84	N	N	N	N	N	P
79	84000	32	123000	34					24	0.9	84	N	N	N	N	N	P
80	24000	49.7	36000	37.4	38000	35.6	50000	97000	34	1.1	58	N	N	N	N	N	P
81	28000	40.2	57000	39	72000	38	112000	126000	32	1		I 202	I	P	P	N	P
82	49000	38.3	44,000	37.6	1,39,000	36			31	1.1	84	I 250	N	P	P	N	P
83	23000	28	58,000	28.7	76000	29	120000		21	0.8	80	N	N	P	P	N	P
84	44000	36.3	61000	41.4	85000	37.7	125000		15	0.7	60	N	N	N	N	N	N

S.NO	R 1	PCV	R2	PCV2	R3	PCV3	R4	R5	UREA	CREAT	SUGAR	LIVER ENZ	BILIRUBIN	IGM	IGG	NSI	WIDAL
85	75000	31.5	79000	30	112000	29	200000		15	0.7	83	N	N	N	N	N	N
86	91000	32	94000	31	114800	28	180000		28	0.9		N	N	N	N	N	N
87	40000	26	43000	25	13000	24	11000	10000	43	0.8	60	N	N	N	N	N	N
88	67000	27	72000	28	45000	25.6	40000	34000	60	1.2	45	N	N	N	N	N	N
89	53000	21.6	1,09,000	21.9					29	0.9	99	N	N	N	N	N	N
90	88000	37.8	130000	36.8	200000	34			34	1.1	128	N	N	N	N	N	N
91	35000	44	26000	43.4	26000	38.7	74000	132000	25	0.8	115	I 200	N	N	N	N	N
92	83000	19.4	78000	19.6					37	0.8		N	N	N	N	N	N
93	9000	5.2	9700	5.6					34	0.9		N	N	N	N	N	N
94	45000	11.8	84000	11.5					20	0.9	81	N	N	N	N	N	N
95	30000	15.6	33000	14					74	1.3	104	N	N	N	N	N	N
96	16000	14.8	17000	15					24	1		N	N	N	N	N	N
97	13000	45.5	16000	33.4	46000				33	1	76	N	N	N	N	N	N
98	9000	29	12000	28	15000	27	34000	50000	30	1	75	N	N	N	N	N	N
99	70000	28	34000	26	32000	20	16000	12000	58	1.4	89	I 560	N	N	N	N	N
100	18000	26	20000	27	34000	28	68000	120000	63	1.4	76	I 300	I	N	N	N	N
101	90000	38	148000	38.4					29	0.6		I 1106	I	N	N	N	N
102	19000	15	12000	14.7	11000	14.4	7000	8700	60	2	68	I 483	I	N	N	N	N
103	79000	24	100000	23.6	120000	24			41	0.9	87	N	N	N	N	N	N
104	13000	44.2	11000	30.1	56000	28.8	62000	112000	24	1	62	I 2500	I	N	N	N	N
105	6000	29.2	12000	28	20000	29.6	22000	24000	22	0.8	62	N	N	N	N	N	N
106	87000	35.1	112000	35.1					27	0.8	118	N	N	N	N	N	N
107	24000	20	20000	19.8	22000	19.6			24	0.9		N	I	N	N	N	N
108	25000	49.2	27000	47	32000	42	65000	100000	30	1	114	I 2500	I	N	N	N	N
109	16,000	50.7	11,000	46.7	17,000	45.8	49,000	96,000	33	1	44	I 800	I	N	N	N	N
110	33000	37.4	40000	29.3	1,79,000	31.5			25	1	68	I 1861	N	N	N	N	N
111	35000	43	119000						117	8	123	I 350	I	N	N	N	N
112	78000	39	77000	36.5	99000	34.5	112000		49	1.2	51	I 395	N	N	N	N	N

S.NO	USGGB	PE	ASCITES	HM	SPM	CXR	TRANS	BLD C/S	PS	MP	MARROW	DIAGNOSIS	OUTC	HEARD	AWARE	TRANS
1	N	N	N	N	N	N	N	NG	N	N	NA	DF	DIS	Y	Y	Y
2	Y	Y	Y	N	N	N	N	NG	MCHC	N	NA	DHF	DIS	N	N	N
3	N	Y	N	N	N	PE	N	NG	MCHC	N	NA	DHF	DIS	Y	N	Y
4	Y	Y	N	Y	N	N	N	NG	NCNC	N	NA	DHF	DIS	Y	N	N
5	N	N	N	N	N	N	10	NG	MCHC	N	NA	DF	DIS	Y	Y	N
6	N	N	N	N	N	N	N	NG	N	N	NA	DF	DIS	N	N	N
7	N	N	N	N	N	N	N	NG	N	N	NA	DF	DIS	N	N	N
8	N	N	N	N	N	N	N	NG	N	N	NA	DF	DIS	Y	Y	N
9	N	N	N	Y	Y	N	N	NG	N	N	NA	DF	DIS	N	N	N
10	N	N	N	Y	Y	BP	N	NG	N	N	NA	DF	DIS	N	N	N
11	N	N	N	N	N	N	N	NG	MCHC	N	NA	DF	DIS	N	N	N
12	N	N	N	N	N	N	25	NG	PANC	N	N	DF	DIS	Y	N	Y
13	N	N	N	N	N	N	N	NG	N	N	NA	DF	DIS	N	N	N
14	N	N	N	N	N	N	N	NG	NCNC	N	NA	DF	DIS	N	N	N
15	Y	N	N	N	N	N	N	NG	N	N	NA	DHF	DIS	Y	Y	N
16	Y	Y	Y	Y	Y	N	N	NG	N	N	NA	DHF	DIS	N	N	N
17	N	N	N	Y	Y	N	N	NG	N	N	NA	DF	DIS	Y	N	Y
18	Y	N	N	Y	Y	N	N	NG	N	N	NA	DHF	DIS	N	N	N
19	Y	Y	Y	N	N	PE	N	NG	N	N	NA	DHF	DIS	Y	N	Y
20	N	N	N	Y	Y	N	N	NG	N	N	NA	DF	DIS	Y	N	Y
21	N	Y	N	N	N	N	N	NG	N	N	NA	DHF	DIS	N	N	N
22	N	Y	N	Y	N	N	N	NG	N	N	NA	DHF	DIS	Y	N	Y
23	Y	N	N	Y	Y	N	N	NG	N	N	NA	DHF	DIS	N	N	N
24	N	N	N	Y	Y	N	N	NG	N	N	NA	DF	DIS	Y	Y	Y
25	N	N	N	Y	N	N	N	NG	MCHC	N	NA	DF	DIS	Y	N	Y
26	N	N	N	Y	Y	N	N	NG	MCHC	N	NA	DF	DIS	N	N	N
27	N	N	N	Y	N	N	N	NG	MCHC	N	NA	DF	DIS	Y	N	Y
28	N	Y	Y	N	N	PE/BP	N	NG	N	N	NA	DHF	DIS	N	N	N

S.NO	USGGB	PE	ASCITES	HM	SPM	CXR	TRANS	BLD C/S	PS	MP	MARROW	DIAGNOSIS	OUTC	HEARD	AWARE	TRANS
29	N	N	N	N	N	N	N	NG	NCNC	N	NA	DF	DIS	Y	N	Y
30	N	N	N	Y	N	N	N	NG	N	N	NA	DF	DIS	Y	Y	Y
31	Y	Y	N	N	N	N	N	NG	MCHC	N	NA	DHF	DIS	Y	N	Y
32	N	N	N	N	N	N	N	NG	N	N	NA	DF	DIS	N	N	N
33	N	N	N	Y	N	N	30	NG	N	N	NA	DF	DIS	N	N	N
34	N	Y	Y	N	N	N	N	NG	N	N	NA	DSS	DIS	N	N	N
35	N	N	N	N	N	N	N	NG	NCNC	N	NA	DF	DIS	N	N	N
36	N	N	N	N	N	N	N	NG	N	N	NA	DF	DIS	Y	Y	N
37	N	Y	N	N	N	N	N	NG	N	N	NA	DHF	DIS	N	N	N
38	N	N	N	N	N	N	N	NG	N	N	NA	DF	DIS	N	N	N
39	N	Y	Y	N	N	N	N	NG	NCNC	N	NA	DHF	DIS	N	N	N
40	Y	Y	Y	N	N	PE	N	NG	N	N	NA	DHF	DIS	Y	Y	Y
41	N	N	Y	N	N	N	N	NG	N	N	NA	DHF	DIS	N	N	N
42	N	N	N	N	N	N	120	NG	N	N	NA	DSS	DEATH	N	N	N
43	N	N	N	N	N	N	N	NG	N	N	NA	DF	DIS	Y	N	Y
44	N	N	N	N	N	N	N	NG	N	N	NA	DF	DIS	N	N	N
45	N	N	N	N	N	N	N	NG	N	N	NA	DF	DIS	N	N	N
46	N	N	N	Y	N	N	N	NG	N	N	NA	DF	DIS	Y	N	Y
47	N	N	N	N	Y	N	N	NG	N	N	NA	DF	DIS	N	N	N
48	Y	Y	N	Y	N	N	N	NG	N	N	NA	DHF	DIS	Y	Y	N
49	N	N	N	N	N	N	30	NG	MCHC	N	NA	DF	DIS	Y	N	Y
50	N	N	N	N	N	N	N	NG	N	N	NA	DF	DIS	N	N	N
51	Y	Y	N	N	N	N	40	NG	MCHC	N	NA	DSS	DIS	N	N	N
52	Y	Y	N	Y	Y	PE	N	NG	N	N	NA	DHF	DIS	Y	Y	Y
53	N	N	N	N	N	N	N	NG	N	N	NA	DF	DIS	N	N	N
54	N	N	N	N	N	N	N	NG	N	N	NA	DF	DIS	N	N	N
55	N	N	N	N	N	N	N	NG	N	N	NA	DF	DIS	Y	N	Y
56	N	N	N	N	N	N	N	NG	N	N	NA	DF	DIS	N	N	N

S.NO	USGGB	PE	ASCITES	HM	SPM	CXR	TRANS	BLD C/S	PS	MP	MARROW	DIAGNOSIS	OUTC	HEARD	AWARE	TRANS
57	N	Y	Y	Y	Y	N	N	NG	MCHC	N	NA	DHF	DIS	N	N	N
58	Y	Y	Y	N	N	N	15	NG	N	N	NA	DSS	DIS	Y	Y	N
59	N	N	N	N	N	N	N	NG	N	N	NA	DF	DIS	N	N	N
60	N	Y	Y	Y	N	N	N	NG	N	N	NA	DHF/CNS	DIS	Y	N	N
61	N	N	N	Y	N	N	N	NG	N	N	NA	E/D	DIS	N	N	N
62	N	N	N	N	N	N	N	NG	N	N	NA	DHF	DIS	N	N	N
63	N	N	N	Y	N	N	90	NG	MCHC	N	NA	DSS	DEATH	N	N	N
64	N	N	N	Y	N	N	30	NG	MCHC	N	NA	DSS/ICH	DEATH	Y	N	N
65	Y	Y	Y	N	N	PE/BP	45	NG	N	N	NA	DSS	DEATH	N	N	N
66	N	Y	N	Y	N	N	N	NG	N	N	NA	DSS	DIS	N	N	N
67	Y	N	N	N	N	N	110	NG	N	N	NA	DSS	DIS	Y	Y	Y
68	Y	Y	N	Y	N	N	N	NG	N	N	NA	ENTERIC	DIS	N	N	N
69	Y	N	N	Y	N	N	N	NG	N	N	NA	ENTERIC	DIS	Y	Y	Y
70	Y	Y	N	Y	Y	PE	N	NG	N	N	NA	ENTERIC	DIS	N	N	N
71	Y	Y	Y	Y	Y	N	N	NG	N	N	NA	ENTERIC	DIS	N	N	N
72	N	N	N	Y	N	N	N	NG	NCNC	N	NA	ENTERIC	DIS	Y	N	N
73	Y	N	Y	Y	N	RLE	N	NG	N	N	NA	ENTERIC	DIS	N	N	N
74	Y	N	N	Y	N	N	N	NG	MCHC	N	NA	ENTERIC	DIS	N	N	N
75	N	N	N	Y	Y	N	30	NG	N	N	NA	ENTERIC	DIS	N	N	N
76	Y	Y	Y	N	N	N	N	CONS	N	N	NA	ENTERIC	DIS	N	N	N
77	Y	N	N	N	N	N	N	NG	N	N	NA	ENTERIC	DIS	N	N	N
78	N	N	N	N	N	N	N	NG	MCHC	N	NA	ENTERIC	DIS	N	N	N
79	N	N	N	Y	Y	N	N	NG	MCHC	N	NA	ENTERIC	DIS	Y	Y	Y
80	N	N	N	Y	N	N	N	NG	N	N	NA	ENTERIC	DIS	N	N	N
81	N	N	N	N	N	N	N	NG	N	N	NA	E/D	DIS	N	N	N
82	Y	N	N	Y	N	N	N	NG	NCNC	N	NA	E/D	DIS	N	N	N
83	N	N	Y	Y	Y	N	N	NG	NCNC	N	NA	E/D/UTI	DIS	N	N	N
84	Y	N	N	N	N	PE	N	KLEBS	N	N	NA	SEPTICEMIA	DIS	N	N	N

S.NO	USGGB	PE	ASCITES	HM	SPM	CXR	TRANS	BLD C/S	PS	MP	MARROW	DIAGNOSIS	OUTC	HEARD	AWARE	TRANS
85	Y	Y	N	Y	Y	BP	N	CONS	MCHC	N	NA	SEPTICEMIA	DIS	Y	N	N
86	N	Y	N	N	N	N	N	PSEUDO	MCHC	N	NA	SEPTICEMIA	DIS	N	N	N
87	N	N	N	N	N	BP	N	PSEUDO	NCNC	N	NA	MENINGITIS	DEATH	Y	N	Y
88	N	N	N	N	N	BP	15	CONS	MCHC	N	NA	SEP SHOCK	DEATH	N	N	N
89	Y	Y	N	Y	Y	N	N	NG	MCHC	P	NA	MALARIA	DIS	Y	N	Y
90	N	N	N	N	N	N	N	NG	N	P	NA	MALARIA	DIS	N	N	N
91	N	Y	Y	Y	N	N	N	NG	N	P	NA	MALARIA	DIS	N	N	N
92	Y	N	Y	N	N	N	20	NG	BLASTS	N	ALL	ALL	REF	N	N	N
93	N	N	N	N	Y	N	N	NG	BLASTS	N	ALL	ALL	REF	Y	N	N
94	N	N	N	Y	Y	N	30	NG	BLASTS	N	ALL	ALL	REF	N	N	N
95	N	N	N	N	Y	N	N	NG	BLASTS	N	ALL	ALL	REF	N	N	N
96	N	N	N	N	N	N	N	NG	BLASTS	N	ALL	ALL	REF	Y	Y	Y
97	N	N	N	N	Y	N	N	NG	RED PLTS	N	MGK	ITP	DIS	Y	N	Y
98	N	N	N	N	N	N	N	NG	RED PLTS	N	MGK	ITP	DIS	N	N	N
99	N	N	N	N	N	N	N	NG	N	N	NA	SNAKE BITE	DEATH	Y	Y	Y
100	N	N	N	N	N	N	55	NG	N	N	NA	SNAKE BITE	DIS	N	N	N
101	N	N	N	Y	N	N	N	NG	N	N	NA	HEPATITIS	DIS	N	N	N
102	N	N	N	N	Y	BP	30	NG	HPH	N	HPH	HPH	DEATH	N	N	N
103	N	N	N	N	N	BP	N	NG	MCHC	N	NA	HIV/OPP I	DIS	Y	Y	N
104	Y	N	N	Y	Y	N	10	NG	MCHC/ATYP LYM	N	NA	LEPTO	DIS	N	N	N
105	N	N	N	N	N	N	N	NG	RED RBC/PLTS	N	APLASIA	A.ANEMIA	REF	N	N	N
106	N	N	N	N	N	N	N	NG	MCHC/ATYP LYM	N	N	LYMPHOMA	REF	Y	Y	N
107	N	N	N	Y	Y	N	N	NG	NUC RBC/HLYSIS	N	PAN	THAL /HS	DIS	N	N	N
108	Y	N	Y	Y	Y	N	N	NG	N	N	NA	UND	DIS	N	N	N
109	N	N	N	N	N	N	105	NG	N	N	N	UND	DIS	Y	Y	Y
110	N	N	N	Y	N	N	35	NG	MCHC	N	N	UND	DIS	N	N	N
111	Y	Y	N	Y	Y	N	N	NG	MCHC	N	NA	UND	DIS	Y	Y	Y
112	N	N	N	N	N	N	N	NG	N	N	NA	UND	DIS	N	N	N

KEY TO MASTER CHART

S.NO	-	SERIAL NUMBER
IP NO	-	INPATIENT NUMBER
WT	-	WEIGHT IN KG
WT FOR AGE	-	IAP GRADING OF MALNUTRITION
N	-	NORMAL
PRE HOSP	-	PRE HOSPITAL TREATMENT RECEIVED
PVT	-	PRIVATE HOSPITAL
GH	-	GOVERNMNENT DISTRICT HQ HOSPITAL
PHC	-	PRIMARY HEALTH CENTRE
ADEQ	-	ADEQUACY OF PRE HOSPITAL TREATMENT
A	-	ADEQUATE
I	-	INADEQUATE
NA	-	NOT APPLICABLE
STAY	-	HOSPITAL STAY IN DAYS
ADM FEVER	-	NO. OF FEBRILE DAYS ON ADMISSION

TOTAL FEVER	-	DURATION FOR WHICH FEVER LASTED
Y	-	YES
N	-	NO
TEND	-	ABDOMINAL TENDERNESS
DIST	-	ABDOMINAL DISTENSION
PUFF FACE	-	FACIAL PUFFINESS
GI	-	GASTROINTESTINAL
P	-	POSITIVE
ERYTH	-	ERYTHEMATOUS RASH
ADE	-	ADVERSE DRUG ERUPTION
AVPU	-	ALERT/VERBAL/PAIN RESPONSIVE/UNRESPONSIVE
PULSE PRES	-	PULSE PRESSURE IN mm Hg
TC	-	TOTAL WBC COUNT
HB	-	HEMOGLOBIN IN GM%
I	-	INCREASED
REF CNT	-	REFERRAL PLATELET COUNT

R1,2,3,4	-	REPEAT PLATELET COUNTS
PCV	-	HEMATOCRIT IN %
CREAT	-	CREATININE
USG GB	-	ULTRASOUND PERICHOLECYSTIC EDEMA
PE	-	PLEURAL EFFUSION
HM	-	ULTRASOUND HEPATOMEGALY
SPM	-	ULTRASOUND SPLENOMEGALY
BP	-	BRONCHOPNEUMONIA
RLE	-	RIGHT LAMELLAR EFFUSION
CXR	-	CHEST X RAY
TRANS	-	TRANSFUSED BLOOD PRODUCT VOLUME IN ML/KG/PATIENT
BLD C/S	-	BLOOD CULTURE AND SENSITIVITY
CONS	-	COAGULASE NEGATIVE STAPHYLOCOCCUS AUREUS
PSEUDO	-	PSEUDOMONAS
KLEBS	-	KLEBSIELLA
PS	-	PERIPHERAL SMEAR

MC	-	MICROCYTIC
HC	-	HYPOCHROMIC
NC	-	NORMOCHROMIC/CYTIC
PANC	-	PANCYTOPENIA
BLASTS	-	LYMPHOBLASTS WITH PROMINENT NUCLEOLI
RED PLTS	-	SEVERE REDUCTION IN THE PLATELET POPULATION
HPH	-	HEMOPHAGOCYTIC HISTIOCYTOSIS
NUC RBC	-	NUCLEATED RBC SUGG OF HEMOLYSIS
ATYP LYM	-	ATYPICAL LYMPHOCYTES
MP	-	PER SMEAR FOR MALARIAL PARASITE
MGK	-	MEGAKARYOCYTE PREDOMINANCE IN THE BONE MARROW
APLASIA	-	RED CELL APLASIA WITH DECREASE IN PLATELETS
DF	-	DENGUE FEVER
DHF	-	DENGUE HEMORRHAGIC FEVER
DSS	-	DENGUE SHOCK SYNDROME
ICH	-	INTRACRANIAL HEMORRHAGE

E/D	-	ENTERIC/DENGUE CO INFECTION
ALL	-	ACUTE LYMPHOBLASTIC LEUKEMIA
OPP I	-	OPPORTUNISTIC INFECTION
LEPTO	-	LEPTOSPIROSIS
A ANEMIA	-	APLASTIC ANEMIA
THAL/HS	-	THALASSEMIA MAJOR/HYPERSPLENISM
UND	-	UNDIAGNOSED
DIS	-	DISCHARGED
REF	-	REFERRED
OUTC	-	OUTCOME
HEARD	-	HEARD THE WORD DENGUE
AWARE	-	AWARE OF SIGNS AND SYMPTOMS OF DISEASE
TRANS	-	AWARE OF TRANSMISSION THRO MOSQUITOES